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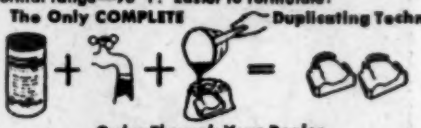

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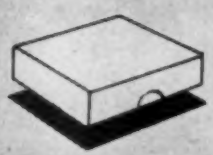

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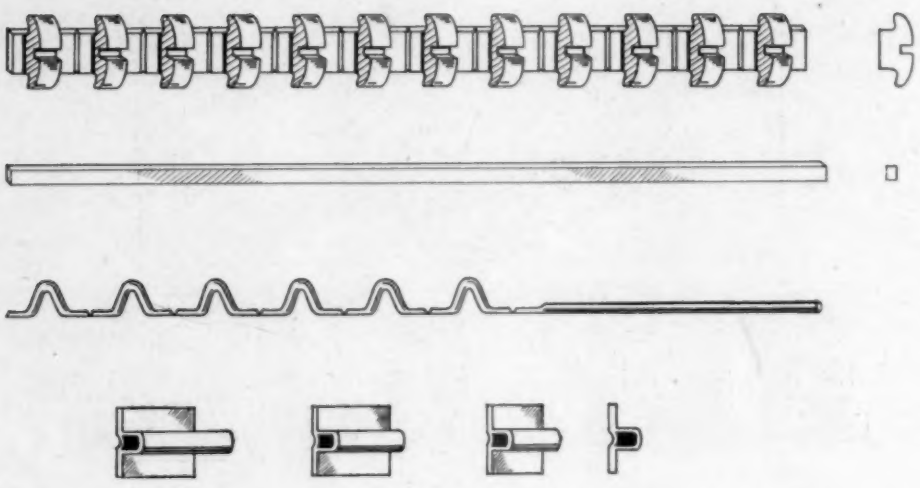

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VOL. 31 SEPTEMBER, 1945 No. 9

Original Articles

ON THE CLASSIFICATION OF DENTOFACIAL ANOMALIES

JOSÉ MAYORAL, M.D., D.D.S.,* BOGOTÁ, COLOMBIA

THE correct classification of dentofacial anomalies is an indispensable condition for making a good diagnosis and, consequently, for planning proper treatment. In a word, it is, in our opinion, the basis of all orthodontics.

This classification should be as full and thorough as possible, even at the risk of sacrificing some simplicity, and should offer us the possibility of including in it, comfortably and rationally, most, if not all, of the cases of dentofacial anomalies that we have to treat. As the adjectives "thorough" and "simple" stand for conditions that it is well-nigh impossible to find together, we are obliged to seek one to the neglect of the other; and here we have chosen exactness rather than simplicity.

Now, the fact is that many of the existing classifications have so resolutely sought this simplicity, which undoubtedly favors their application, that they take into account only a limited number of dentofacial anomalies. And so we have, for instance, classifications that are concerned only with the anomalies of occlusion, while others deal exclusively with those of the jaws. But to be complete, a classification must reckon the anomalies of the soft parts as well as those of the jaws, of the teeth, and of the temporomandibular joints, and, of course, those which have to do with occlusion.

The classification of dentofacial anomalies should not be confused with the means and methods of diagnosis. For though a good classification presupposes the practice of exact diagnosis, it should not be subordinate to it. Let it be noted that a correct knowledge and, in consequence, a correct classification of dentofacial anomalies are to be attained through gnathostatic models, profile roentgenograms, x-ray photographs, photostatic pictures, Atkinson's key ridge, and other means of diagnosis.

Anomalies are not diseases but may be a consequence of disease or a perturbation of development. At the same time they differ from lesions in that

*Professor of Orthodontics at the Faculty of Dentistry, National University of Colombia, Bogotá.

there is never any longer a reaction of the organism. Now, dentofacial anomalies may be divided, according to their seat, into five groups which are here given in the order that it is most convenient to follow in clinical practice for making a diagnosis. They are:

1. *Anomalies of the soft parts,* comprising the lips, cheeks, tongue, the floor of the mouth, soft palate, masticatory muscles, and the circulatory and nervous systems of the mouth.

2. *Anomalies of the jaws,* considered both by themselves and in relation to the rest of the head and the planes of the body. (Here the chief anomalies are those of the dental arches, or, rather, the alveolar processes and their teeth.)

3. *Anomalies of the teeth,* studied both as isolated organs and in relation to the normal curve of the arches, the sagittal plane, and the plane of occlusion.

4. *Anomalies of the temporomaxillary joints,* which include alterations in the condyles and limitations in their movements.

5. *Anomalies of occlusion,* or malocclusions, which have to do with the mutual or reciprocal situation of the teeth of one and the other arch, when so placed as to obtain the greatest number of points of dental contact. (The anomalies of this group do not really constitute an independent pathologic entity having its own etiology and pathogeny, but are always secondary, being the consequence of one or more of the anomalies indicated in the other four groups.)

Once the seat of the anomaly is known, its classification ought to follow. The anomalies of the first four groups, which have a pathologic individuality, are by Nature herself divided into three subgroups, according to the following concepts:

1. *Time* or evolution—whenever a certain form, normal in a given period of the individual's growth or of the development of the organs, either persists into a later period, or, on the contrary, appears before the due time.

2. *Space*—whenever the space occupied by the organs is not normal; which may be due to some irregularity of position or of placing or of the seat itself of a given organ; or to abnormality in the *direction* or orientation of a given part in respect of the planes and lines of the body; or to variations in *volume,* or size; or to alterations of *forms,* whether macroscopic, or microscopic.

3. *Number*—whenever the organs or their parts are more in number or fewer than they ought to be.

From the point of view of cause and duration, of etiology and pathogeny, there are *congenital anomalies and acquired anomalies, or deformities.* Both are the result of disturbing causes acting on the subject, on the gametes from which he himself has come, or on his ancestors, in such a way as to produce hereditary alterations. Now, the earlier the action of the disturbing cause on the individual—whether such action be in the embryonic stage or on the fetus, on the child or on the adult—the greater will be the resulting anomaly. Furthermore, we find a cause of anomaly or deformity in everything which disturbs the development or the morphologic and functional normality of the soft parts of the mouth, the jaws, the teeth themselves, and the temporomandibular articulations, especially the whole system of stresses and strains to which each tooth is subjected, the result of which is that the tooth is constantly being pushed in every direction by equal and contrary forces.

We have said that after diagnosing the seat and nature of each anomaly, it is necessary to determine with precision the disturbing cause (etiology) and its action, and also the morbid, abnormal concatenation of observable alteration (pathogeny).

Congenital anomalies of the mouth are morphologic irregularities having their origin during the intrauterine development and postnatal evolution of the dental follicles. They are, that is to say, such anomalies as are produced in the period of the embryonic growth of the individual and his organs, and are, as a matter of fact, already established at the moment of birth, and they include those anomalies in the form of the teeth which appear at teething.

Acquired anomalies, or deformities, are alterations produced when the embryonic development of the individual and his organs is already finished.

What we have said so far about the classification of dentofacial anomalies may be summed up in Table I.

TABLE I

	ACCORDING TO PLACE	ACCORDING TO NATURE		ACCORDING TO PATHOGENY
DENTOFACIAL ANOMALIES	1. Soft parts 2. Jaws 3. Teeth 4. Temporomandibular joints 5. Occlusion: secondary, following the preceding	Time		
		Space	position direction volume shape	Congenital
		Number	increased decreased	Acquired

Congenital anomalies, or anomalies in development, may have already been observed in the patient's family, in which case the cause is hereditary. Acquired anomalies, or deformities, are not inherited.

The classification of dentofacial anomalies, as outlined in the foregoing table, points out first of all the seat, or place in which the anomaly is observed, next the nature or class of the alteration, and lastly its pathogeny and etiology. This is the order that must be followed in the clinic to arrive at a diagnosis, and this is the procedure that must serve as a basis for the solution of the problem of orthodontic terminology. Without a full classification of anomalies, it is impossible to choose a proper terminology, or to appreciate, criticize, and interpret any terminology that has so far been put forward or that may later on be proposed.

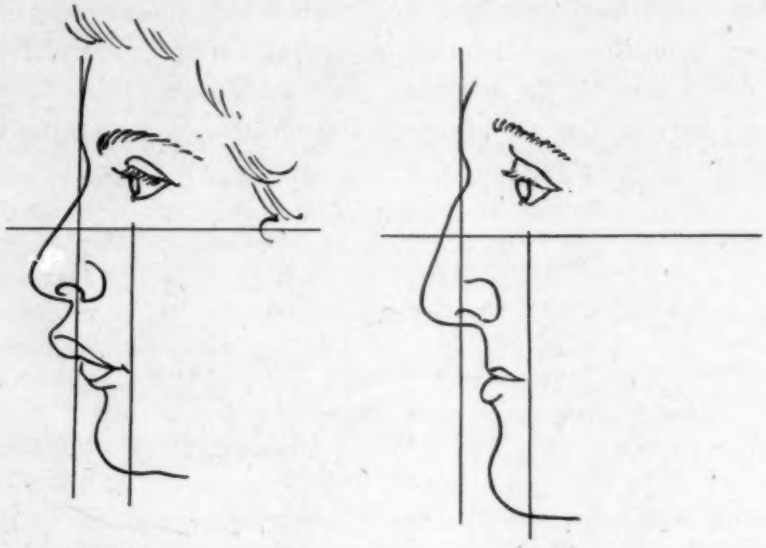
FIRST GROUP. ANOMALIES OF THE SOFT PARTS

These are subdivided into anomalies of time, of space, and of number. Those of time are always congenital, the most important being facial clefts, or prosoposchisis (Greek, *prosopon,* face, and *schisis,* division); labial clefts, or cheiloschisis (Greek, *cheilos,* lip); and uvular clefts, or staphyloschisis (Greek *staphyle,* bunch of grapes, hence, "uvula").

Those of space are anomalies of position, or situation, of direction, of volume or size, and of form. Many of them generally accompany the anomalies of the jaws; such, for instance, are procheilia (Greek and Latin, *pro,* in front, before), that is, abnormally protruding lip (Fig. 1, *A*); retrocheilia (Latin, *retro,* back), or abnormally receding lip (Fig. 1, *B*); laterocheilia (Latin, *latus, lateris,* side), a deviation of a lip to one side or another. Among the anomalies of position and direction we must also include laterostomia, or deviation of the buccal orifice to one side or another of the saggital plane; lateroglossia, deviations of the tongue; laterostaphylia, deviations of the uvula; and glossoptosis, a falling of the base of the tongue.

The following are anomalies of volume and shape: enlargement of the buccal orifice, macrostomia (Greek, *makrós,* large and *stoma,* mouth); lessening of the buccal orifice, microstomia (Greek, *mikrós,* small); increase in the size of the

lip, macrocheilia; decrease in the size of the lip, microcheilia; hypertrophy of the labial frenum, or adherence of the lip to the gum, synechiecheilia (from Greek, *syneché*, joining); increase in the bulk of the cheek, or macromalia (Latin, *malae*, cheek), and the contrasting decrease, or micromalia; increase in the bulk of the tongue, macroglossia, and the contrasting decrease, microglossia.



A. *B.*

Fig. 1.—Example of anomalies of soft parts. *A*, Procheilia. *B*, Retrocheilia.

TABLE II

- GROUP I. ANOMALIES AND DEFORMITIES OF THE SOFT PARTS
  - Time
    - Prosoposchisis: facial clefts
    - Cheiloschisis: labial clefts
    - Staphyloschisis: uvular clefts
    - Glossoschisis: tongue clefts
  - Space
    - Position and Direction
      - Procheilia: lip more in front than normal
      - Retrocheilia: lip behind normal position
      - Laterocheilia: deviations of lips to one side or the other
      - Laterostomia: deviations of buccal orifice to one side or the other of sagittal plane
      - Lateroglossia: deviations of tongue
      - Glossoptosis: falling of base of tongue
      - Laterostaphylia: deviations of uvula
    - Volume and Shape
      - Macrostomia: large buccal orifice
      - Microstomia: small buccal orifice
      - Macrocheilia: big lip
      - Microcheilia: little lip
      - Synechiecheilia: labial frenum hypertrophy
      - Macromalia: large cheek
      - Micromalia: small cheek
      - Macroglossia: big tongue
      - Microglossia: little tongue
      - Synechioglossia: lingual frenum hypertrophy
  - Number
    - Increased
    - Decreased

We must likewise consider, as one of the most important anomalies of the soft parts, the lack of stretching power, or tone (Greek, *tonos*, stretching, straining), in the facial muscles, particularly the orbicular muscle of the lips, a phenomenon that often accompanies those maxillodental anomalies caused by buccal respiration.

The most important anomalies of the soft parts may be summed up in Table II.

SECOND GROUP. ANOMALIES OF THE JAWS

In this group, the anomalies of time are those clefts which, during the development of the face, separate the embryonic sprouts. When their seat is the jaws, they come under the heading of gnathoschisis (Greek, *gnathos*, jaw, and *schizo*, to cleave), and when the cleft is in the hard palate, it is called uranoschisis (Greek, *ouranós*, roof-sky, hence "palate").

Those of position and direction are deviations of the jaws from the position that they ought normally to occupy with relation to the planes of the body. When the deviation is toward the middle or sagittal plane (Fig. 2, *A*), it is

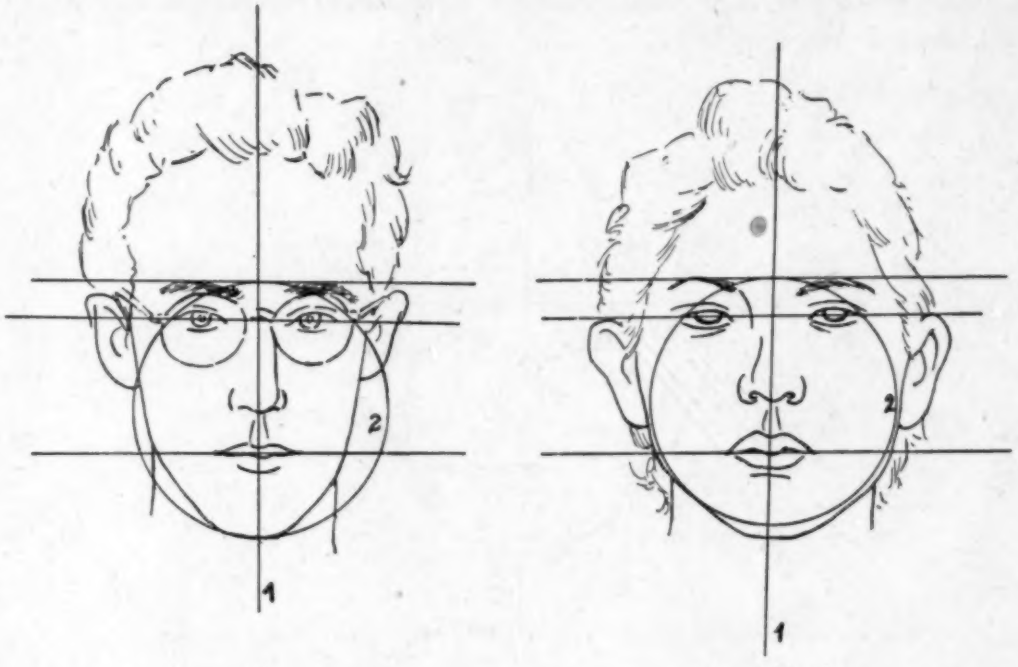


Fig. 2.—*A*, Endognathism: upper and lower. *B*, Exognathism: upper and lower. *1*, Sagittal plane. *2*, Mayoral's circumference, with center at spinal point and radius to glabella, ought to pass through the two zigia (sing., zigion), the two gonia (sing., gonion) and the gnathion, in normal persons.

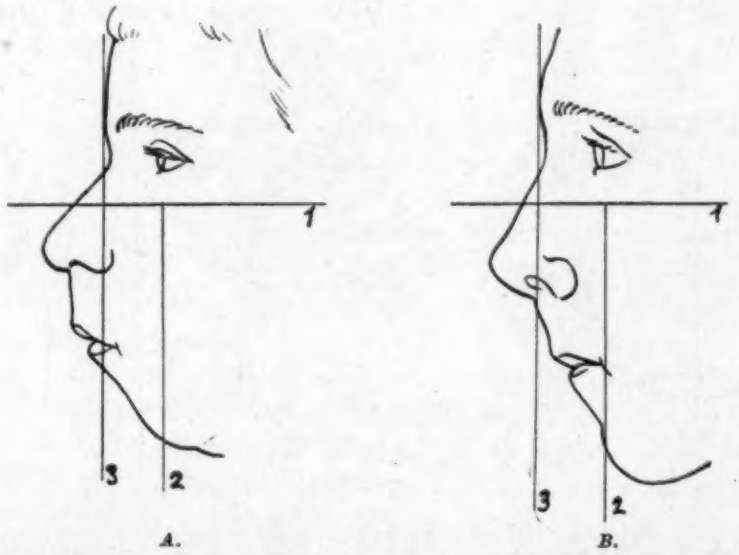


Fig. 3.—*A*, Upper, prognathism. *B*, Lower retrognathism. *1*, Francfort's plane. *2*, Simon's plane. *3*, Izard's plane, perpendicular to Francfort's and passing through the glabella.

known as endognathism (Greek, *endom*, within), and when it is away from the sagittal plane (Fig. 2, *B*), it is called exognathism (Greek, *ektós*, out). Either may be upper and lower, unilateral or bilateral. When the jaws deviate forward, we have prognathism (Fig. 3, *A*); when backward, retrognathism (Fig. 3, *B*). Both prognathism and retrognathism may be upper and lower, and total or simply alveolar, according to how much is out of place—the whole jaw or only the alveolar processes.

Maxillary deviations to one side or the other of the sagittal plane are termed laterognathism (Fig. 4), which may be either dextrognathism—toward the right, or levognathism—toward the left. The chin, quite independently of the position of the jaw, may deviate forward—progeneism (Greek, *géneion*, chin), or backward—retrogeneism.

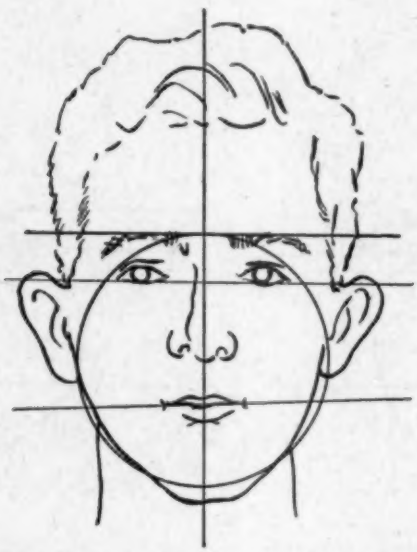


Fig. 4.—Lower levognathism. (Deviation of chin to left of sagittal plane.)

Fig. 5.

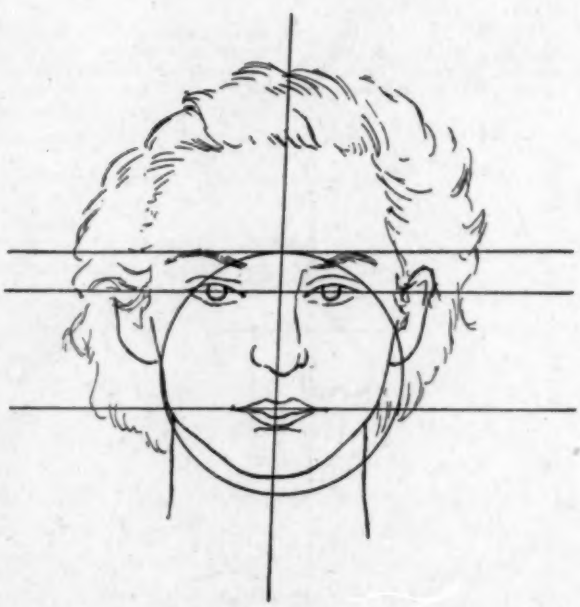


Fig. 6

Fig. 5.—Macrognathism. (Distance from spinal point to glabella is less than from spinal point to gnathion.)

Fig. 6.—Micrognathism. (Distance from spinal point to glabella is greater than from spinal point to gnathion.)

The anomalies of size and shape are increase in volume, or macrognathia (Fig. 5), and decrease in volume, or micrognathia (Fig. 6), both of which may be double or single, upper or lower. Very interesting is any variation in the shape of the lower jaw, such as hypergonia (Greek, *gonía*, angle), which is an increase in the size of the angle, and hypogonia, which is a decrease in the same. Anomalies of number, which are very rare indeed, are classified as polygnathia, when more than two jaws occur, and agnathia, when there is a lack of jaw.

The anomalies of the jaws are summed up in Table III.

TABLE III

- **GROUP II. ANOMALIES AND DEFORMITIES OF THE JAWS**
  - **Time**
    - Gnathoschisis: clefts in either jaw
    - Uranoschisis: clefts in bony palate
  - **Space**
    - **Position and Direction**
      - Endognathism (upper, lower) — jaws narrower than normal
      - Exognathism (upper, lower) — jaws wider than normal
      - Laterognathism (levognathism, dextrognathism) — jaws twisted with middle part to one side of sagittal plane
      - Prognathism (upper, lower; total, alveolar) — jaws more in front than normal
      - Retrognathism (upper, lower; total, alveolar) — jaws behind normal position
      - Progeneism: chin more in front than normal
      - Retrogeneism: chin behind normal position
    - **Volume and Shape**
      - Micrognathism (double, upper, lower) — deficiency in size of jaw
      - Macrognathism (double, upper, lower) — excess in size of jaw
      - Hypergonia: excess in extension of angle of lower jaw
      - Hypogonia: deficiency in extension on angle of lower jaw
  - **Number**
    - Polygnathia: more than two jaws
    - Agnathia: lack of jaw

THIRD GROUP. ANOMALIES OF THE TEETH

Anomalies of time or evolution are due to the coming out of temporary or permanent teeth either before or after the proper time, or to the falling out of the temporary teeth at an unseasonable period.

Anomalies of space may have to do with position. Such, for instance, is transposition, or double migration (Latin *migrare*, to move from one place to another). We have simple migration when a tooth is not in its right place, though not far from it. In speaking of these phenomena, we use the trunk-word, or stem, "gression" (Latin, *gradi*, to walk, step, move), to which we add some prefix to indicate the specific direction of the movement or separation from the normal position.

"Gression," then, may take place on a horizontal or a vertical plane. On the horizontal plane the tooth may be displaced toward the labial or buccal region, producing what is known as labiogression or buccogression; or toward the tongue, in which case we have linguogression; or in the direction of the mesial part of the mouth, forming a mesiogression; or toward the distal region, when it is called distogression (Fig. 7). On the vertical plane, gression may tend toward the thickness of the bone, to produce ingression (Latin *in*, in or

into); or toward the outside of the gum, forming an egression (Latin, *ex*, outside, out of). (See Fig. 8.) When we find a gression that does not strictly follow any of the six principal directions just mentioned, but rather some intermediate line or trend, we have to use a compound term to indicate the main lines of abnormal position between which the tooth tends. Thus, the term *mesiolinguogression* would indicate a deviation of the tooth in an intermediate direction comprised by the lingual and mesial regions (Fig. 9).

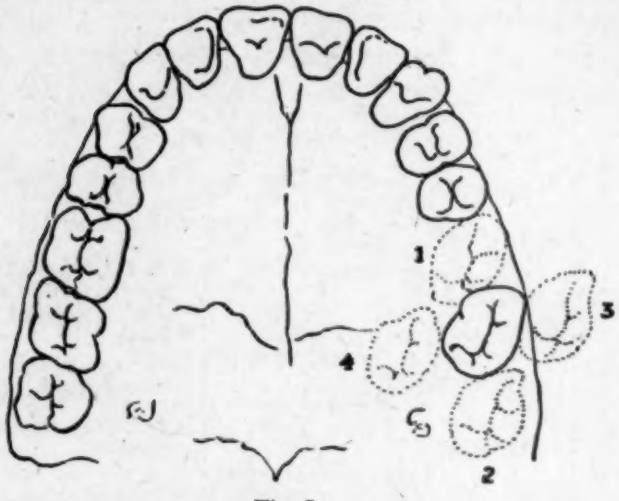


Fig. 7.

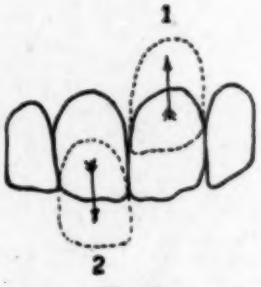


Fig. 8.

Fig. 7.—*1*, Mesiogression. *2*, Distogression. *3*, Labiogression, or Buccogression. *4*, Linguogression.

Fig. 8.—*1*, Ingression. *2*, Egression.

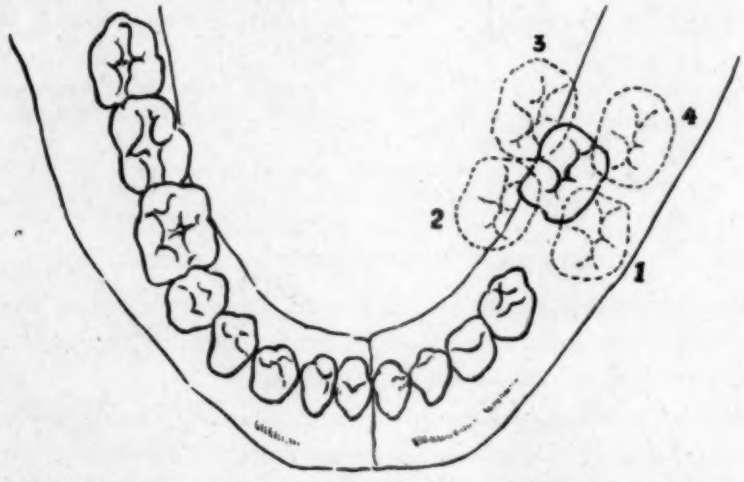


Fig. 9.—*1*, Mesiobuccogression. *2*, Mesiolinguogression. *3*, Distolinguogression. *4*, Distobuccogression.

When the teeth are outside the alveolar processes, we have dental aberration (from Latin, *aberrare*, to wander away, to stray).

Anomalies of direction may be observed in teeth that are rooted in their proper place as well as in those that are not. The tooth may be regarded as if it had turned about an axis—any of its axes, in fact—in such a way as to change its trend or orientation in respect to the chief planes of the body.

When a tooth seems to have moved about its vertical axis, we have rotation (Latin, *rota*, wheel). When we may imagine the tooth as having turned about a horizontal axis, we have an anomaly known as radical version (Latin, *vertere*, to turn).

Rotations may be mesiovestibular—that is, mesiobuccal or mesiolabial—mesiolingual, distovestibular and distolingual, according to whether the deviation of the mesial and distal surfaces be toward the outside or toward the inside (Fig. 10).

Versions, which, be it noted, are produced when a tooth turns round a horizontal axis, are denominated vestibuloversion—that is, labioversion or buccoversion—linguoversion, mesioversion, and distoversion, according to whether the trend or inclination of the tooth be toward the vestibular region or the lingual or the mesial or the distal (Fig. 11).

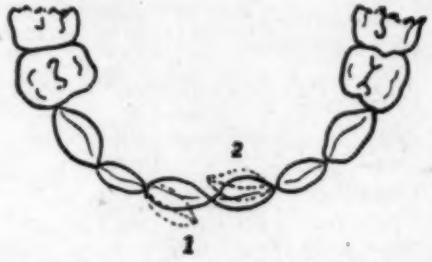


Fig. 10.—*1*, Mesiolabiorotation. *2*, Mesiolinguorotation.

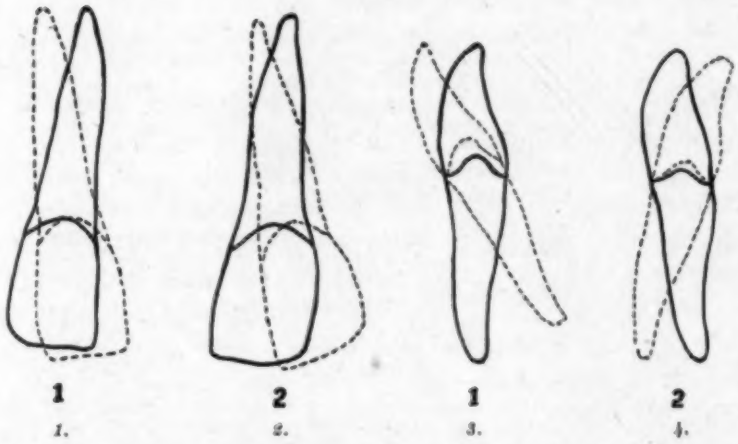


Fig. 11.—*1*, Mesioversion. *2*, Distoversion. *3*, Labioversion. *4*, Linguoversion.

The anomalies in the size, or volume, of the teeth are those of excess, macrodontia, and those of deficiency, microdontia. These anomalies may affect the whole of the tooth or be confined to the crown or the root.

The chief anomalies in shape are as follows: an excess in the number of cusps or roots; bent or twisted roots; conoid teeth; hypoplasia of the enamel, and the condition known as Hutchinson's tooth.

Anomalies of number consist of an excess or deficiency in the number of teeth that the individual ought to have at a given age. A variation in this respect may be due to the presence of supernumerary teeth, to a delay in the falling out of temporary teeth, to the existence of enclosed teeth, to the premature loss of teeth, or to a lack or insufficiency in dental development.

The anomalies of the teeth are summed up in Table IV.

FOURTH GROUP. ANOMALIES OF THE TEMPOROMAXILLARY ARTICULATIONS

As alternations in the temporomaxillary joints play a very important part in producing other dentofacial anomalies, we are obliged to make a diagnosis and classification of them.

TABLE IV

GROUP III. ANOMALIES OF THE TEETH

- **Time**
  - **Eruption**
    - precocious — temporary or permanent teeth
    - tardy — temporary or permanent teeth
  - **Fall**
    - precocious — temporary teeth
    - tardy — temporary teeth
- **Space**
  - **Position**
    - Double migration or transposition: teeth that exchange position
    - Simple migration; gressions
      - horizontal (mixed)
        - vestibulogression
        - linguogression
        - mesiogression
        - distogression
      - vertical (mixed)
        - ingression
        - egression
    - Diastema: separation of teeth that should be together
    - Aberration
      - heterobuccal: out of the mouth
      - buccal: out of the gums
  - **Direction**
    - Versions
      - vestibuloversion
      - linguoversion
      - mesioversion
      - distoversion
    - Rotations
      - mesiovestibular
      - mesiolingual
      - distovestibular
      - distolingual
  - **Size**
    - Macrodontia
      - total
      - crown
      - root
    - Microdontia
      - total
      - crown
      - root
  - **Shape**
    - Geminodontia: union of one or more teeth
    - Disjunctodontia: separation of parts of the same tooth
    - Increase in number of cuspids
    - Increase in number of roots
    - Bent, twisted and divergent roots
    - Conoid teeth
    - Stains and hypoplasia of enamel
    - Hutchinson's tooth
- **Number**
  - Increased
    - by persistence of temporary teeth
    - by presence of supernumerary teeth
  - Diminished
    - by lack of development
    - by premature loss

TABLE V

GROUP IV. ANOMALIES OF TEMPOROMANDIBULAR JOINTS

- Absence or limitations of normal movement or mandibular constriction
  - With abnormal position of the condyles or condylism
  - Without condylism, with normal position of the condyles
- Condylisms; loss of normality of relations between the condyles and the glenoid fossae
  - Procondylism
  - Retrocondylism
  - Laterocondylism
    - levocondylism
    - dextrocondylism
- Inocclusion or adaknia: inability to close mouth completely, lack of contact between upper and lower teeth

The anomalies of the temporomandibular, or temporomaxillary, joints are, in fact, an absence or limitation of normal movement owing to constriction of the jaw (retraction of the muscular fiber, ankylosis, etc). The loss of normal relations between the condyle and the glenoid cavity gives rise to anomalous positions of the former, known as condylisms. When the condyles are situated in front of the place that is rightly theirs in the position of normal occlusion, we say that there is procondylism, a condition in which—be it noted—the jaw is projected forward and a lower prognathism occurs. When the jaw deviates toward one side, as a result of the wrong position of the condyles, we say that there is laterocondylism, which may be toward the right, dextrocondylism, or toward the left, levocondylism. In these cases there will be lower laterognathism. Lastly, when the jaws do not quite close, that is, do not come into perfect

contact with each other, we say there is inocclusion or adaknia (Greek, *daknó*, to bite).

Table V is a synthesis of the anomalies of the temporomaxillary articulations.

FIFTH GROUP. ANOMALIES OF OCCLUSION

Normal occlusion is a resultant of various factors, which may be put into four groups, according to the following considerations:

1. Normality of the soft parts of the mouth.
2. Normality of the jaws.
3. Normality of the teeth.
4. Normality of the temporomandibular articulations and of the movement of the jaw on these joints.

From all this it is clear that the anomalies of occlusion are a result of the phenomena we have described in the foregoing four groups of dentofacial anomalies.

In reality, the anomalies of occlusion are not a single entity; and what we observe, in the soft parts, in the teeth and in the temporomaxillary joints, is a number of irregularities which necessarily alter the normal reciprocal situation of the upper and the lower teeth when these are placed in the position of maximum contact.

In grouping the anomalies of occlusion, we have followed Angle's classification, which is so well known that we have not thought it necessary to give it here. The defects that have been imputed to it are here corrected, since before coming to these particular irregularities we have clearly classified the dentofacial anomalies in the foregoing four groups: to wit, those of the soft parts, those of the jaws, those of teeth, and those of the temporomaxillary articulations.

This classification of anomalies, which, at first sight, might seem extremely complex, is not so in reality. The students of the Facultad de Odontología (School of Dentistry) of Bogotá soon become accustomed to making use of it in clinical work, and it has been found that the orthodontic records drawn up in accordance with this classification make its application simple and practical. The fact is that all the cases of dentofacial anomalies that crop up in practice may be classified in this way.

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CARRERA 9
21-68

TOWARD A WORKING CONCEPT OF PHYSICAL GROWTH

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SEVERAL years ago, the writer published a preliminary compilation of the ways in which different authors, in viewing the child from the standpoint of his anatomic and physiologic characteristics, used the words "growth and development." To this disconcerting, yet thought-provoking, compilation, was appended a proposed "concept of physical growth" and an invitation to other workers "to evaluate the concept and to elaborate it or cite its weaknesses."[1]

A number of readers subjected the paper to critical appraisal and made valuable suggestions. Also, from the writer's continuing participation in teaching and research there were discovered needed extensions of the concept. In rewriting the paper for publication in this JOURNAL, it is intended to bring it abreast of the writer's current thought and have it come to the attention of those human biologists particularly concerned with the calcification and eruption of teeth and with craniofacial and faciodental interrelationships.

THE PROBLEM

For upwards of a decade the writer has been engaged in investigation and instruction relating to what he terms "The Physical Growth of the Child." During this time he has recurrently found himself confronted with literature in which an attempt is made to distinguish between growth and development or growth and maturation. On the one hand, the practice of trying to give these words differential meaning appears to have become deeply intrenched. On the other hand, the published efforts at differentiation are frequently lacking in clarity, are often inconsistent, and almost without exception fall far short of delineating the full scope of the age to age changes actually taking place in the child's body.

In question form, the problem may be cast as follows: What is physical growth? Is it satisfactorily defined as cell multiplication or as increase in body size? What is physical development? Is there a meaningful and fruitful distinction between growth and development? Can physical development be adequately defined as increase in structural complexity or as progressive cellular specialization? What is physical maturation? Is it possible and helpful to circumscribe the characteristics of maturation as distinguished from those of growth and development? Can physical maturation be appropriately defined as encompassing changes in the complexity of physiologic functions, in the texture of body tissues, in the alignment of body parts, or in the proportional relations between parts?

To secure information on these questions one may turn to direct attempts at definition or to the use of the words "growth," "development," and "maturation" in research papers. The discussion which follows will draw upon both approaches.

GROWTH AND/OR DEVELOPMENT AND/OR MATURATION

A committee report of the White House Conference on Child Health and Protection states: "We use the two terms, growth and development, advisedly

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as there is a useful and significant distinction between them."[2] Gesell, on the other hand, is no less emphatic in support of the opposite position. He writes, ". . . it is unnecessary, and even undesirable, to insist upon a distinction between growth and development. We shall use these terms interchangeably."[3] A recent statement by Macy is appropriate to epitomize the situation as it stands: "There is little agreement upon nomenclature among the scientists investigating the question of 'child development' . . . Even the terms 'growth' and 'development' have not acquired definition and differentiation acceptable to the entire group."[4]

Definitions of Growth.—In a paper on the topic, "What Is Growth" Frank states, ". . . *growth* means simply the process by which one cell undergoes self-multiplication, the result being that two cells are generated or produced."[5] This concept of growth as cell multiplication is countered by Davenport as follows: "Growth, as it has been defined by biologists for the past fifty years, is increase in size."[6] The definition given by Marriott and Jeans constitutes a merging of the positions of Frank and Davenport. To wit.: "Physical growth may be defined as the increase in the quantity of living tissue of the body, either by increase in the size of cells already present or by the development of new cells."[7]

While the foregoing definitions limit physical growth to "increase" in size or in number of cells, other definitions use the more inclusive term "change" in place of increase. Wetzel writes, "We prefer . . . to conceive of growth as associated with a *change* in size rather than to specify that growth consists exclusively of an increase in size."[8] Bakwin and Bakwin state: "Growth . . . is strictly a quantitative concept and refers, in the physical sphere, to change in number or size of units."[9]

Concepts of growth more comprehensive than any of the above are held by Breckenridge and Vincent, by Hammett, and by Krogman. Hammett's view is that growth represents "the combined expression of developmental and incremental factors. Increase in cell number is as much growth as is increase in cell size. Increase in cellular specialization is as much growth as is increase in cellular segregation."[10] According to Krogman, "Physical growth per se is . . . increase in size, change in proportion, and progressive complexity."[11] Breckenridge and Vincent write: "Growth is both quantitative and qualitative. By the term 'growth' we are implying two aspects of change which take place—'growing' and 'growing up.' "[12] "Growing" is identified by these authors as "increase in size," and "growing up" as "changing in structure and improving in function."

Definitions of Development.—Greulich defines development as "becoming progressively more complex . . . growing in complexity."[13] In like vein, the previously mentioned committee report of the White House Conference on Child Health and Protection states that "development implies an increase in complexity, such as we see in the formation of the four-chambered heart of the infant from the simple pulsating tube of the embryo."[2] Bakwin and Bakwin take the position that to define development as increase in complexity is "unsatisfactory for several reasons."[9] With particular reference to use of the word "increase," rather than "change," they write: "It is questionable whether the fully developed heart is more complex than the fetal heart."

There are a group of definitions which have the one characteristic in common that they regard development as applying to changes other than increases in magnitude. According to Lussier, "Development is progress toward maturity

. . . it implies progressive maturity; not increase in dimensions."[14] The view of Draper, Dupertius, and Caughey is that development "connotes the continuous modification of an individual's total life processes which transpire between the egg stage and adult form. It deals not with size but with events of functional rearrangement and especially with the time at which these events occur, and their relation to one another in respect to the time of their occurrence."[15] Stuart writes: "Just as mankind has slowly developed through the ages, so each individual man must start as a single fertilized cell and must pass through a great variety and number of stages before he reaches maturity. . . . The term *development* properly refers to this process of change from the single cell to the complex mature adult, in contrast to *growth* which refers to simple increase in size."[16]

Working definitions adopted by Macy and by Goldstein are as follows. For Macy: "We have used development to indicate progress in the specialization, organization, or differentiation of tissues, function, or the entire organism."[4] For Goldstein: "Development is here used in the sense of proportional differentiation, which may be followed by means of indices."[17] Apparently representing a blend of these concepts is a statement from Stuart: "If a child merely developed, man and child would be alike in size, though very different indeed in capacity and proportions."[16]

Definitions of development which encompass changes in the size of the organism are incorporated in the writings of Davenport, Hurlock, and Wetzel. Hurlock writes, ". . . physical development is not limited to changes in size. The child is not merely a 'miniature adult' . . . his whole body shows proportions different from those of the adult."[18] According to Davenport, "Increase in size, combined with differentiation, or the process of specialization of parts, constitutes development."[6] It is Wetzel's view: "Growth is that fundamental property of living things (organisms) concerned only with their change in size or number, as distinguished from the co-relative property of *differentiation* which has to do with changes in kind or type of structure. Acting together, growth and differentiation result in *development*."[19]

The concept of Frank is that "development really consists of three important processes; first, growth which forms new cells by self-multiplication of preceding cells; second, differentiation of these cells as regards both structure and action (i.e., specialization as bone, muscle, nerve, blood, and other cells); and third, the organization of all the parts which growth and differentiation have produced."[5]

Definitions of Maturation.—In a special treatment of the "meaning of maturation" by Todd, the word is considered to symbolize all of the inevitable changes (exclusive of augmentations in size) which an organism undergoes during its life cycle. To quote: "During childhood we grow: that is we increase in dimensions. . . . But we also grow up . . . grow older and . . . grow old. This business of growing up, growing older, and growing old is quite different from that of growing: it implies progressive maturity, not increased dimensions. . . . There is, in the concept of progressive maturity, the implication of its inevitability."[20] Unfortunately, Todd did not discuss the biologic considerations which led him to regard such changes as those of complexity, form, and positional arrangement as components of "progressive maturity" and to set aside changes in size as belonging in a "quite different" category. It is of interest to note that in another publication by Todd the statement is made: "Growth . . . the increment in bodily size or bulk . . . is one expression of physical development or the growing-up process."[21]

Gesell envisages maturation as "an active physiological process" which constitutes a portion of growth and development. He writes, "Maturation is the intrinsic component of development (or of growth) which determines the primary morphogenesis and variabilities of the life-cycle. Although the word growth is sometimes loosely used as synonymous with maturation, the former is the more comprehensive term including all the developmental differentiations of the organism in response to external as well as internal environments."[8]

By Krogman and by Bakwin and Bakwin maturation is regarded as identical with development. In a general discussion of "Principles of Human Growth," Krogman writes: "We'll return to two themes again and again: the first is growth, the second is development—size and maturation."[11] Bakwin and Bakwin state: "The terms development and maturation, as here used, refer to the innumerable stages and processes through which human form, function and behavior pass from conception to maturity. . . . Growth on the other hand . . . refers . . . to change in number or size of units."[9] They add, "Development is innately determined and can be influenced by the environment to only a minor degree. . . . Growth is susceptible to environmental influence." The view of Bakwin and Bakwin that "maturation takes place according to a plan and a sequence which are innate" is similar to Todd's position that maturation denotes "change or progress over which no external influence has any power."[20] In this connection it is pertinent to raise such questions as: Is there any less "inevitability" about the number of pedal digits or the size of the external ear than about the eruption of deciduous and permanent incisors? Is the appearance of axillary and pubic hair, or union of metacarpal epiphyses with diaphyses, any more "innately determined" than occurrence of the adolescent acceleration in leg length or in chest circumference?

Many writers use the term "maturation" with a highly specific connotation. Not infrequently the word is employed as equivalent to (a) the reduction division of germ cells, (b) the advent of the menarche, (c) the myelination of nerve fibers, or (d) the sequence of structural change characteristic of blood cells. It will suffice to give an example of one of these restricted uses. Pryor writes, ". . . each germ cell, male and female, undergoes a series of peculiar divisions, during which the number of chromosomes is reduced by half. This process is called 'reduction division' or 'maturation.' "[22]

Inconsistencies in Definition.—It follows from the materials presented above that when different attempts to define "growth," "development," and "maturation" are placed in juxtaposition they are neither harmonious for any of the three terms taken singly, nor distinguishing for any two of the terms considered together.

Essentially the same definition is given by Lussier for development and by Todd for maturation. Hammett's concept of growth is seemingly identical with the concept of development presented by Davenport. Gesell regards growth and development as synonymous terms. Bakwin and Bakwin employ development and maturation interchangeably. For Frank, growth is a subdivision of development; for Breckenridge and Vincent, development is a subdivision of growth; for Gesell, maturation is a subdivision of growth and development.

The same item of structural change—the first few cell divisions following fertilization—is used by Frank as an illustration of growth (i.e., cell multiplication) and in the White House Conference report as an illustration of development (i.e., increase in complexity). As an example of growth: ". . .

growth begins in the egg . . . this cell becomes separated into two cells. . . . These two then separate again into four, into eight, into sixteen. . . .''[5] As an instance of development: ''It is possible in many instances to have considerable development with very little growth in size. This obviously occurs during the first days following the fertilization of the ovum. . . .''[2] The discrepancy, of course, arises primarily from the differences in definition and secondarily from the fact that during the early cleavage stage there is cell multiplication without increase in the size of the organism. By giving consideration to a different item—change in amount of voluntary musculature during childhood—another aspect of the incongruities in definition is revealed. Change in quantity of musculature during the childhood years is due to accretions in cell size rather than to increase in cell number. Accordingly, under the concepts of the White House Conference report it constitutes an example of growth (i.e., increase in size) and under the concepts of Frank an example of lack of growth (i.e., absence of self-multiplication of cells).

Possibly the most widespread of the attempted differentiations between growth and development is that which identifies growth with ''increase in size'' and development with ''increase in complexity.'' Some champion these definitions as ''useful and significant,'' while others decry them as ''unsatisfactory.'' Clearly the definitions cannot be interpreted as encompassing all of the anatomic and physiologic changes which occur during either the prenatal or postnatal portions of the human life cycle. They disregard instances of decrease, both in size and complexity, and they fail to take cognizance of whole areas of change, such as those of form or shape and of position or alignment.

The Area of Operations.—What is the situation when one turns from the available attempts at definition to the specific utility of the terms ''growth,'' ''development,'' and ''maturation'' in research? Are the three words employed differentially and found functionally useful in planning, executing, or reporting research? Or, do differences in use fail to manifest themselves?

In the same sense that one investigator uses ''growth progress,'' another uses ''progression of development,'' and a third uses ''progress in maturation.'' To identically the same succession of changes one investigation applies the term ''growth trend,'' a second the term ''developmental pattern,'' and a third the term ''maturation sequence.'' One investigator employs ''level of growth'' in the same way that another employs ''developmental status,'' and a third, ''stage of maturation.'' In the same sense that ''segment of growth'' is used in one study, ''phase of development'' is used in a second, and ''period of maturation'' in a third. The writer has in his files a thesis on osseous *growth*,[23] a monograph on osseous *development*,[24] and a book on skeletal *maturation*.[20] All three studies deal with the same problem and employ the same type of data.

Certainly those who formulate and direct research programs do not find it practicable to distinguish between problems of growth, problems of development, and problems of maturation. Nor do those responsible for preparing systematic reviews or syntheses of the reasearch literature find it feasible and fruitful to subdivide their materials according to these three categories.

Many authors employing the phrase ''growth and development'' appear to do so without intending each of the words conjoined to carry a separate meaning. Apparently in similar manner use is sometimes made of the phrases ''development and maturation'' and ''growth and maturation.'' Frequently one finds various combinations of the terms used in contexts which imply their complete interchangeability. In this connection it will suffice to cite a study by Baldwin, Busby, and Garside, affording two reciprocal statements of purpose in

successive paragraphs. In one paragraph it is stated that the objective of the study is "to trace and analyze specific traits of growth" to discover "fundamental principles of development."[25] In the other paragraph the aim is alternately given as that of observing and studying items of "development" to discover "basic principles of growth." Obviously the substitute use of growth and development by these investigators is solely for variety of language.

It appears that no "useful and significant distinction" has yet been drawn between the physical growth of the child, the physical development of the child, and the physical maturation of the child. Consequently, in the opinion of the writer, it is justifiable to conclude that the three terms are appropriately regarded (at least for the present) as equal in scope and as fully interchangeable. From the standpoint of economy in writing and printing, "physical growth" has the advantage of being the shortest of the three terms. For some years, both in manuscript preparation and in the classroom, the writer has used this term almost exclusively.

PROPOSED DEFINITION

What is physical growth? It is *the sequence of somatic modification which a biologic organism undergoes during its ontogenetic life history.* More explicitly, and with particular reference to the subclass of mammals that includes the human organism, physical growth may be defined as *the entire series of anatomic and physiologic changes taking place between the beginning of prenatal life and the close of senility.*

Basically, these definitions emanate from a twofold objective: To delimit the sector of science that students of physical growth investigate, and to embrace both its explored and unexplored areas. This objective will be seen to be realized by the restriction of physical growth to somatic (anatomic and physiologic) changes, and by the identification of physical growth with nothing less than the whole gamut of such changes.

Operating under the proposed general concept, the remainder of the paper will be given over to presenting a suggested working classification of the kinds of anatomic or structural change currently known to occur during human ontogeny. No attempt will be made at this time to present a parallel systematization covering the diversity of presently known physiologic changes.

The structural or anatomic changes thus far investigated may be grouped into changes in complexity, changes in size, changes in shape or form, changes in position, and changes in texture and pigmentation. This grouping, like all empirically derived schemes of classification, should be approached not as something final and binding, but as a schema perhaps sufficiently comprehensive and meaningful to merit adoption pending a more adequate proposal. While the writer has found the schema as itemized to be highly useful both in teaching and research, it is possible that other students of physical growth would find it more convenient to subdivide the investigated changes somewhat differently. There is also the probability that future scientists will discover additional phases of structural change. Notwithstanding, it is necessary to face the immediate need of moving "toward a working concept of physical growth"—a concept that can be both generally stated and satisfactorily particularized. From the standpoint of particularization, it appears fruitful, at least tentatively, to regard anatomic growth as pertaining to changes with advancing age in the complexity, size, form, positional arrangement, texture, or pigmentation of any structural unit of the organism and of the organism as a whole.

Changes in Complexity.—The human body undergoes a variegated succession of changes in its structural complexity. There is cell multiplication and cell resorption, tissue differentiation and tissue obliteration, organ emergence and organ submergence. Gill arches appear and disappear, hair is acquired and lost, teeth individuate and are discarded. There are differences in the timing of complexity changes from part to part. Moreover, most units register appreciable alteration with age in direction of change, while many show complete reversal of direction. As a consequence, from practically every segment of the life cycle it is possible to draw examples of increases in complexity, plateaus in complexity, and decreases in complexity.

The precursor of the human child is a single cell, the fertilized ovum. Cell division commences almost immediately, and by two or three days after fertilization the ovum consists of eight to sixteen cells. One of these cells probably constitutes the nascent child. Certainly by the end of the first week there has formed a small cluster of cells which represent the anlage of the child proper. At birth the child's body is estimated to consist of two hundred billion cells, and at the close of adolescence of twenty-six trillion cells. In making this transition from a unicellular organism to a multicellular organism of some twenty-six trillion cells, the child passes through a circuitous process of cell formation and cell dissolution. As early as the latter half of the first prenatal month, pronephric ("first kidney") cells are being resorbed, and, by the latter half of the second month, cartilaginous cells of the clavicle and maxilla are undergoing dissolution and replacement by bone cells. During the late fetal and early postnatal weeks there is considerable degeneration of connective tissue cells in the chamber of the middle ear. In childhood and adolescence there is reduction in the number of germ cells, production and destruction of bone cells, manufacture and discard of blood and skin cells. The nerve cells comprising the cortex increase during the prenatal period, remain constant during childhood and early adulthood, and become reduced in number in old age.

Early in the second week following fertilization the human organism is a simple disc consisting of one kind of tissue—ectoderm. In rapid succession the organism becomes a bilaminar plate (with the appearance of endoderm tissue) and a trilaminar embryonic shield (with the appearance of mesoderm tissue). During the latter half of the first month and the first half of the second month there occur three inpocketings of ectoderm from which are formed markedly different derivative tissues. The first gives rise to the nervous system, the second to the lens of the eye, and the third to the enamel of the teeth. Later derivatives of ectoderm include nails and hair. Derivatives of mesoderm arising during the latter half of the first month include vascular tissue and blood, connective tissue and cartilage. Later products are osseous and gonadal tissues, dentine and cementum. What is the theme? Tissues appear and tissues disappear. In the embryonic period, ectoderm is replaced by ectodermal derivatives and mesoderm by mesodermal derivatives. In the fetal and infancy periods, the mesenchymal membrane (sclerogenous connective tissue) of the cranium is replaced by osseous tissue. At all ages from the second prenatal month into early adulthood substitutions are taking place in the skeleton of osseous tissue for cartilaginous tissue.

Changes in complexity occur not only from the standpoint of cells and tissues but also from the standpoint of body organs and systems and segments and features. The organism passes, in turn, from an embryonic shield of three germ layers to an embryo with articulate head and trunk, brain and heart, noto-

chord and gut; from an embryo without arms or legs, nose or ears, bones or teeth, fingers or toes, eyelids or nails, to a fetus manifesting these structures; from a newborn infant lacking erupted teeth, lacking ossification of the carpal bones and of most epiphyses, lacking calcification of the permanent teeth, and lacking the presence of coarse axillary and pubic hair, to an adult with all these items of complexity. Again, however, the trend is not one of cumulative increase in complexity. More nearly it is one of continual interplay of increase and decrease. This may be illustrated in the embryo, in the fetus, in childhood, and in adolescence.

In the embryo: The shoulder region of the human embryo at one month following fertilization, with its rudimentary limb buds, is manifestly more complex than the shoulder region of the embryo one week younger, which gives no indications of the upper limb buds. Similarly, the upper extremities of the embryo at two months, with their evident demarcations of arm, forearm, palm, and finger regions, register a clear increase in complexity relative to the undifferentiated limb buds of one month earlier. On the other hand, the young embryo acquires a series of branchial or gill arches in the neck region, a pair of ridges extending down the trunk from axilla to groin, and an external tail. These items of complexity are not retained. The gill arches and furrows are made over into a varied array of facial and cervical structures, the lower two-thirds of the so-called "milk lines" are obliterated (the pectoral portions give rise to the mammae), and there is loss of the external tail.

In the fetus: During the third and fourth months so-called manal "touch pads" are acquired and lost. These small mounds of flesh, eighteen in number, appear at the tip of each digit and at the base of each cleft between digits. Between the fourth month and birth a coat of body hair (lanugo) is acquired and shed, the permanent teeth anlagen remain largely unchanged in complexity, while the deciduous teeth pass from ectodermal tooth buds to complex structures characterized by such ectodermal and mesodermal derivatives as enamel, dentine, pulp, and cementum.

In childhood: The child's dentition undergoes marked increase in complexity during the early postnatal years and decided decrease in complexity during later childhood. This fact is vividly portrayed by placing in juxtaposition the dentition of the child at three selected ages. The dentition of the typical neonate comprises twenty deciduous teeth almost completely calcified, no teeth erupted into the oral cavity, and no calcification of the permanent teeth. About 5 years of age the child's dentition consists of twenty deciduous teeth fully calcified and erupted plus twenty-eight permanent teeth in varying stages of calcification. By 14 years of age the entire deciduous complement has been discarded and the complexity of the dentition reduced to twenty-eight permanent teeth fully calcified and erupted plus four permanent third molars partially calcified and unerupted.

In adolescence: Distinguishing changes in complexity during adolescence include (a) appearance of coarse, pigmented hairs in the pubic and axillary regions and (b) marked reduction in the number of bone masses constituting the skeleton. It has been estimated that around puberty the human skeleton consists of about three hundred and fifty bone masses. By obliteration of the cartilaginous plates separating epiphyseal bones from diaphyseal bones, the skeleton of the young adult is reduced to roughly two hundred bones. (It should not be inferred that the process of skeleton simplification is restricted to the adolescent period. While union of epiphyses with diaphyses is the predominant

feature of skeletal growth during this peroid, coalescence of bone masses characterizes almost the entire life cycle. In the terminology of Krogman, "we 'lose' some 600 bones or bone centers from fetal through adult life."[11])

Examples of decreasing complexity for the periods of maturity and senility are to be found in gradual obliteration of the cranial sutures, in loss of head hair, and in reduction of number of permanent teeth.

Changes in Size.—Human growth is characterized by a wide variety of changes in size. This variety accrues not only from differences in rate of change, but also from differences in duration and in direction of change.

Patterns of change in size vary in duration from less than a day for some components of the body to practically the entire life cycle for others. In the case of a specified cell of the embryonic disc, changes in size are confined to the *hours* from its initial existence as a separate cell to its division into two daughter cells. With reference to a given tubule of the mesonephros ("middle kidney"), changes in size can cover only the few *weeks* between its appearance and disappearance. In the case of a deciduous central incisor, patterns of change in size are restricted to the *years* (approximately seven) from differentiation of the bud to shedding. With reference to the overall magnitude of the heart, or brain, patterns of change can extend over *decades*—over almost the entire life cycle.

Patterns of change in size are rarely trends of linear increase. Predominantly they are trends which change in slope (alter their direction) with age. In some instances the alterations in slope scatter over the full gamut from rapid acceleration, through no change, to rapid retardation. In other instances the alterations lie within the more restricted zone of gradations between no change (plateau) and rapid acceleration (steep rise).

Reversals in the direction of magnitude trends are occurring throughout life. In the embryo the external tail increases in length during the fifth and sixth weeks and decreases in length during the seventh and eighth weeks. During the fifth and sixth weeks there is also increase in size of the mesonephric ducts (which are taken over as the male sex ducts) and the Müllerian ducts (which become sex ducts in the female). In the early fetal period there is decrease of the Müllerian ducts in males and the mesonephric ducts in females. In the older male fetus the gubernaculum (a ligament extending from the caudal end of the testis to the floor of the scrotum) increases in length through the sixth month, shortens 50 per cent during the seventh month and, by birth, becomes reduced to about one-fourth its earlier maximum. Body weight increases during the prenatal period, decreases about 6 per cent during the first few days following birth, and increases again during infancy. The weight of the suprarenal glands increases during the fetal period, decreases about 50 per cent during the first two weeks after birth, and remains constant during infancy. A fibrous pad of adipose tissue in the region of the instep increases in size during the fetal period (obscuring the arch of the foot) and decreases in size during the first two to three postnatal years. The thickness of the subcutaneous adipose tissue on the thorax increases during the late fetal and early postnatal months and decreases between about six months after birth and 6 years of age. The deciduous incisors increase in length during the late fetal and early postnatal months, remain on a plateau between 2 and 5 years of age, and decrease in length (as a result of root resorption) prior to being shed. Other deciduous teeth show this same pattern of ascent, plateau, and descent except that their durations are longer and the timings later. In males, the trend for thickness of the subcutaneous adipose tissue on the arm is one of moderate increase between 6 and 11 years

of age and of marked decrease between 11 and 16 years. The typical trend for weight of the thymus gland is one of ascent up to about 12 years of age and decline over the years from 12 to 25. For the thyroid gland, the weight trend shows regular increase from birth to adolescence, no appreciable change during the adult years, and decrease in senility. Stature increases at varying rates over the first two decades of life, remains stationary for the four succeeding decades, and decreases in old age; weight of the liver follows a similar trend. The cortex of the brain increases at a slowing rate over the first fifteen years, shows no appreciable change in magnitude from 15 to 60 years, and then undergoes considerable reduction (the corpus callosum shrinking to less than half its maximum size).

Length of the uterus increases during the fetal period, decreases during the first fortnight after birth, remains practically stationary during infancy and early childhood, increases again during late childhood and adolescence, has another fairly stationary period, and then another period of decrease following the menopause. The pharyngeal tonsil (adenoid mass) increases in size from early infancy through the third postnatal year, maintains a constant magnitude during childhood, and gradually undergoes recession after adolescence. On the whole the thickness of the skin increases from infancy up to young adulthood and then decreases as age advances—in many areas the reversal in trend is such that the skin becomes as thin in senility as it was in infancy. The alveolar processes increase in height preceding and during the eruption of teeth, remain at a fairly stationary height over most of the period they are supporting teeth (with some localized fluctuations paralleling the loss of deciduous teeth and their replacement by permanent teeth), and then decrease markedly following loss of the permanent teeth.

Patterns of change in size which vary between the limits of steep rise and plateau, like those that also have descending phases, are variously timed and ordered. The pattern for change in size of the ovum is one of plateau (no increase) during the first few days after fertilization, followed by ascent (rapid increase). For the bones of the middle and inner ear the pattern is one of increase in size in the fetal period and stationary size throughout the postnatal period. In the case of the first permanent molars there is increase in size during infancy and early childhood, followed by no change in size after about 9 years. For males, the anteroposterior diameter of the larynx increases during infancy, remains constant during childhood, increases again during adolescence, and again passes to a period of constancy. Similarly, the size of the penis and scrotum in males and of the mammae in females increases but slightly over the childhood years, shows marked increase during the adolescent years, and approximates a plateau over the succeeding twenty-five years. The width of the head increases rapidly during infancy, much more slowly during childhood and adolescence, and not at all during adulthood and senility. The weight of the lens of the eye increases at a slowing rate up to 3 years of age and at a constant rate thereafter (i.e., the trend ascends in a curve convex on age during infancy and early childhood and continues as a slowly rising linear trend throughout late childhood, adolescence, adulthood, and senility). The trend for weight of the pituitary gland increases fairly regularly from birth to adolescence. A fairly common pattern for the period from birth through adolescence is that of rapid increase during infancy, slow increase during childhood, and rapid increase again in the second decade—this is found for surface area of the body, weight of the kidneys, length of the upper and lower extremities, width of the shoulders and hips, girth of the arms, legs, and chest. The trend for size of the

third permanent molar approximates a plateau during infancy and early childhood, rises during the age period from about 8 to 22 years, and follows along a plateau thereafter.

When a body structure changes its size, it may increase or decrease equally throughout, it may increase or decrease more in one direction than another, it may increase in one dimension while decreasing in another, or it may increase in one area, remain constant in a second area, and decrease in a third. In the prenatal period, between about six and nine weeks of age, the mesonephros ("middle kidney") is continuously increasing in size in one region and decreasing in size in another. Succinctly, there is a wavelike progressive reduction of its more cranial tubules and augmentation of its more caudal tubules. Throughout childhood many of the changes in bone size represent a concerted process of bone increase and decrease. The shaft of the femur changes in its cross-sectional area by external accretion and internal resorption. Such is the interplay of increase at the periphery and medullary resorption that the shaft of a newborn may be placed inside the medullary cavity of an adult. The mandible changes in its width by increases on the outer surface and by decreases along its lingual wall. Similarly, it changes in its depth by bone deposition along the posterior edge of the ramus and bone absorption on the anterior edge. From the beginning of the second decade on, the parenchymatous components (medulla and cortex) of the thymus gland decrease in size while the connective tissue and fat component increases.

Changes in size may or may not synchronize with changes in cell number. This is to be expected since some cells maintain a constant size (e.g., bone cells), some cells change in size (a marked example is found in the increase in length of the axon portion of certain peripheral nerve cells), and for some tissues there are intercellular changes (e.g., adipose tissue). Drawing upon the materials already cited, it will suffice to bring together two contrasting illustrations of asynchronism and one illustration of synchronism. During the first few days after fertilization there is cell multiplication without increase in size. In the latter part of the prenatal period and the first decade of postnatal life there is increase in the size of the nervous system in the absence of production of nerve cells. At all ages—wherever osseous changes occur—increase and decrease in bone mass is a direct function of increase and decrease in bone cells.

Changes in Shape.—It has been amply shown that the human organism undergoes ontogenetic changes in size and in complexity. We now turn to a third aspect of structural change—change in shape (i.e., in form, contour, proportion, configuration). Included among the ontogenetic trends thus far investigated on the human organism are an extensive series of trends of change in shape or form. Whether viewed from the standpoint of general body proportions, or of the configuration for specific segments and organs, the embryo differs from the neonate, the neonate from the adolescent, and the adolescent from the adult.

Changes in form are brought about in many ways. Thompson has suggested that certain internal modifications are analagous with the alterations made by the glassblower. To quote: "The alimentary canal, the arterial system including the heart, the central nervous system . . . all begin as simple tubular structures. And with them Nature does just what the glassblower does. . . . For she can expand the tube here and narrow it there; blow off a lateral offshoot or caecal diverticulum; bend the tube, or twist and coil it; and infold or

crimp its walls. . . . Such a form as that of the human stomach may be regarded as an ill-blown bubble, a bubble that has been rendered lopsided by a trammel or restraint along one side."[26] Modifications in external body form are known to be brought about, singly or in combination, through changes in complexity, changes in position, and changes in size. The shape of the germinal disc is changed by unequal rates of cell multiplication in different regions. The shape of the embryo's trunk is changed by appearance of the limb buds and by disappearance of the external tail—also, of course, by changes in the number, size, and positional arrangement of its internal components. The shape of the fetal thorax is changed by differential changes in size, by the downward tilting of the ribs, and by other changes in the disposition of its contents. Similarly, the shape of the face during childhood is changed by eruption of the teeth and by varying rates of dimensional change.

Procedures commonly employed in the study of changes in shape or form are (a) juxtaposition or superimposition either of pictorial representations of body structures or of the structures themselves, and (b) computation and graphing of indices derived from measurements taken either on the body or on pictorial representations thereof. Illustrations of procedure *a* are as follows: Specimens of the skull for the late embryonic period, infancy, childhood, adulthood, and senility may be placed in juxtaposition and attention directed to their differences in form. Full-length photographs of the child at a few widely separated ages may be reduced to one height and then serially aligned in order to portray changes in body proportions. Lateral silhouettes at some half-dozen infancy and childhood ages may be arranged side by side for the purpose of exhibiting modifications with age in the protrusion of the abdomen, or in the curvature of the posterior aspect of the body stem. Changes in facial contour during infancy and childhood may be studied by superimposing tracings of profile photographs of the head and face obtained, say, shortly after birth and at the ages of 1, 3, 6, 10, and 15 years. Similarly, progressive changes in the configuration of particular bones, or internal organs, may be followed by superimposition of a small number of serial roentgenogram tracings. Contour drawings of the foot, or of some other part, may be secured at a few selected ages, all reduced to the same length (or width), and then superimposed to show the trend of variation in shape.

From these illustrations it will be seen that the closely allied methods of juxtaposition and superimposition yield pictorial results that are readily interpreted. Unfortunately, however, the fruitful application of these methods is limited, i.e., they find their principal usefulness where trends are considered sufficiently indicated by displaying less than a dozen spaced stages. For purposes in which sequences of change in shape are to be studied using data at frequent consecutive ages over a fairly long segment of the life cycle, a more adequate method is that of computing and graphing ratios or indices—procedure *b* above. While index numbers and trends are somewhat less readily interpreted, being numerical and graphic rather than pictorial, they have the clear advantage of permitting more exhaustive representation and intercomparison of patterns of change.

In its simplest form a ratio expresses one measurement of the body in relation to another measurement. The two measurements may be dimensions, areas, volumes, or weights. Assume that in early infancy the length of the body stem (head and trunk) is 14 inches and the length of the lower limbs 7 inches. One may compute either the ratio of the former to the latter (14/7)

and obtain an index of 2.0, or the ratio of the latter to the former (7/14) and obtain an index of 0.5. The index 2.0 would be read as indicating a stem twice (200 per cent) as long as the lower limbs, and the index 0.5 as indicating lower limbs one-half (50 per cent) as long as the stem. An extensive series of indices have been used in the study of changes in shape. While the complete list would make tedious reading, the following may be mentioned as representative: the cephalic index (ratio of head breadth to head length), the facial index (ratio of face length to face breadth), the nasal index (ratio of nose breadth to nose length), the palatal index (ratio of palate breadth to palate length), the thoracic index (ratio of thorax width to thorax depth), the hip-shoulder index (ratio of hip width to shoulder width), the skelic index (ratio of length of lower limbs to stem length), the intermembral index (ratio of length of upper limbs to length of lower limbs), the brachial index (ratio of forearm length to arm length), the crural index (ratio of leg length to thigh length), the hand index (ratio of hand breadth to hand length), and the foot index (ratio of foot breadth to foot length). In using any index to investigate a trend of form change, the procedure is that of computing the index at successive ages and of plotting the resulting values on a graph. It should be recognized that change in an index trend in a given direction may be produced by any one of several combinations of directional change in the underlying size trends. For example, a descending index trend may accrue from (a) one dimension—the numerator—increasing less rapidly than the other, (b) one dimension remaining constant while the other increases, (c) one dimension decreasing while the other remains constant, or (d) one dimension decreasing more rapidly than the other.

Studies have been made of age-to-age changes both in the shape of given parts and in the proportionate relationships between parts. Two weeks after fertilization the human organism has the form of an ovoidal plate or disc, predominantly composed of nascent head region. By five weeks the organism is cylindroid rather than platelike, its head is flexed forward and downward, its facial region is broad and short (breadth practically twice length), and its thorax is deep and narrow (depth more than one and one-half times width). At eight weeks the organism is less strongly curved; its nose is short and broad (length one-half width); its thorax is circular (equal in width and depth); its head width is one and one-half times shoulder width, two and one-half times hip width, and one and one-half times lower limb length; and its lower limb length is equal to shoulder width, three-fourths upper limb length and one-seventh stature. Compared with the body shape of the young adult, the neonate has a long stem with short limbs, a low-bridged nose and compressed face, a more protruding abdomen, less concave cervical and lumbar flexures, more nearly equal girths of head and thorax, and less elongated hands and ears.

The proportionate contribution to the total organism of the head component decreases throughout the first two decades of the life cycle. Expressed in percentage of stature, length of head and neck declines from approximately 65 for the early embryo, through 40 for the young fetus and 25 for the one-year-old infant, to 19 for the young adolescent. Expressed in percentage of chest girth, head girth decreases from around 140 in the young fetus, through 100 in late infancy, to less than 70 in early adulthood. Expressed in percentage of hip width, head width declines from approximately 250 for the eight-weeks embryo, through 125 at birth and 80 four years after birth, to less than 55 in early adulthood.

Index trends depicting the change in shape of different aspects of the head and face follow a variety of courses. The cephalic index decreases markedly during prenatal life (head width being 95 per cent of length in the early embryo and about 80 per cent at parturition), rises during early infancy, falls between about 1 and 3 years, remains almost constant between 3 and 5 years, and registers a slight decrease during late childhood and adolescence (for young adults of northwest European ancestry the index approximates the index at birth). The facial index increases during the late embryonic and early fetal period (from less than 60 to around 77), remains constant during the latter half of prenatal life, increases during childhood and adolescence, remains constant during adulthood, and declines in senility. The trend for the nasal index (white race) declines rapidly over the period from eight prenatal weeks (200) through birth (100) to about six years (65), continues a gradual decline throughout childhood, remains on a plateau over the adult years and, for males, shows a slight rise in old age.

The thoracic index increases from less than 60 in the early embryo to 115 at the beginning of the fetal period, remains constant throughout the fetal period, and then increases to around 135 during the first five postnatal years. The hip-shoulder index gives a rising trend during fetal life and the first three postnatal months, a downward trend between 3 months and about 1 year, and a rising trend from 1 to 4 years. The ratio of lower limb length to trunk length increases from the appearance of the limb buds in the fifth week through 37 at eight weeks to 87 by the middle of the prenatal period, declines slightly during the latter half of the prenatal period, increases to practically 150 by 12 years of age, then decreases to less than 145 in males and 140 in females.

For the brachial index there is a rise during fetal life, a fall during the first postnatal year, a plateau between 1 and 5 years, a second fall to about 12 years, and a second rise from 13 to 16 years. For the crural index the trend ascends during late childhood and descends during adolescence. The hand index decreases from around 100 in the sixth week, through approximately 60 at two months and 50 at birth, to about 45 in adulthood. The foot index declines to 50 at two months and 40 at birth, with only a small further decline postnatally.

The stomach begins as a swelling of one portion of the digestive tube and passes from this capsular form through a varied sequence of form modifications to a J-shape—the shape which is characteristic for the period from 12 years on through adult life. The heart begins as a tube which is first modified into a simple spiralled-S, and then rapidly reshaped through a fairly complex process of differential looping, swelling, and merging. Postnatally the heart tends to become more pyramidal in shape, i.e., broader at the base and more pointed at the apex. During early adolescence, the aperture of the larynx in the male undergoes anteroposterior elongation and becomes strongly elliptical, while, in the female, the superior pelvic aperture decreases in ellipticity and becomes nearly circular.

Changes in Position.—Besides changing in size and shape and complexity, anatomic parts change their positions. The known shifts in position for different organs, segments, and other body components combine to yield a composite view of changes in all directions. The lungs, diaphragm, and liver migrate downward; the kidneys and ears migrate upward. There is a backward twisting of the upper extremities and a forward torsion of the lower extremities. The

stomach rotates about its long axis clockwise and tilts to an oblique position across the abdomen from left to right; the heart rotates counterclockwise and assumes a somewhat oblique position in the thorax from right to left. The eyes move toward the center of the face. Nerves migrate out to the muscles and skin. There is tilting of the ribs, bending of the feet, turning of the ovaries. The teeth show rotation and movements in both the vertical and horizontal planes. Even the hard palate "descends by addition below and absorption above."[27]

Positional changes for the heart include caudodorsad migration (movement downward and backward), tilting, and rotation. Initially the site of the heart is chiefly ventral to the lower portion of the head region. By the fifth week it is located in the area in front of the primitive cervical vertebrae. Two weeks later its position is considerably more dorsal, and it lies partly opposite the lower cervical vertebrae and partly opposite the upper thoracic vertebrae. By the middle of prenatal life it has moved down in front of the third to eighth thoracic vertebrae, shifted away from verticality to an oblique alignment from right to left, rotated forward so that part of its earlier ventral surface has become caudal, and turned laterally bringing its earlier right side more to the front. At birth the orientation of the heart is more transverse than in late childhood. During the first five postnatal years there is a slight tilting back toward the vertical—a tilting registered by the movement of the apex (most caudal and left lateral portion of heart) from the level of the eighth vertebra down to the level of the ninth or tenth vertebra. In late maturity the heart moves to a somewhat more lateral and more horizontal placement in the thorax.

When the stomach is first identifiable as a spindle-shaped enlargement of one portion of the gut, it lies approximately in a vertical position and is only slightly below the level of the lung buds. In the course of the ensuing month it comes to be located at an appreciably lower level, rotates 90 degrees about its long axis, and assumes a generally oblique position from left to right across the abdomen. (The more caudal displacement of the stomach in this period is appropriately interpreted as positional rearrangement—a shift in setting—rather than as a "descent." There are a number of such realignments in the early embryo where it is not known whether the main direction of change is actively caudad, or only apparently caudad as a consequence of cephalad movement of neighboring parts.) During later prenatal life the stomach gradually moves to a more horizontal orientation, and in early infancy it lies almost transversely in the abdomen. Over the childhood years the pyloric end of the stomach moves down, so that in early childhood the organ shifts through varying degrees of obliquity and in late childhood it returns to a nearly vertical position.

The upper limb buds are at first directed downward, the lower limb buds ventrocaudad. Soon all four limbs extend outward almost at right angles to the sides of the trunk. They then bend so that, for the upper limbs, the palmar surfaces of the nascent hands face the thorax while the elbows project outward and slightly caudad, and, for the lower limbs, the plantar surfaces of the nascent feet face the abdomen while the knees project outward and somewhat cephalad. Subsequently, (a) both pairs of limbs undergo a torsion of 90 degrees about their long axes, (b) the thumbs shift proximally, (c) the long axes of the feet turn through 90 degrees, and (d) there is adduction and rotation of the great toes. The feet lie initially along the same axes as the shafts of the legs. About the seventh week they begin to turn out of the leg planes

and approach their eventual right-angle relationship with the legs. When the great toes are first individuated, each points away from the four other toes and has its plantar surface turned toward them; slowly each moves to a position parallel with the other toes and rotates sufficiently to bring the plantar surface into the sole plane. The thumb is at first attached to the palm near the plane of the base of the index finger; gradually it shifts proximally along the lateral side of the palm. Regarding the 90-degree torsion of limbs, this takes place in contrasting directions. For the upper limbs there is a twisting at the shoulders so that the elbows are rotated from their outward position backward. For the lower limbs there is a turning at the hips so that the knees are brought forward. Full frontal alignment of the knees is not attained until after birth. The typical neonatal orientation of the lower limbs is one of divergent thighs, flexion at the knees, and soles practically facing each other. Only gradually during infancy are the limbs extended, the knees brought side by side, and the soles turned downward.

The primordia of the eyes are located near the center of the ventral surface of the forebrain. From this earliest site the two optic outgrowths diverge and the eyes become laterally placed. About the middle of the second prenatal month the axes of the primitive eyes are separated at an angle of 160 to 180 degrees. In the third month the angle is reduced to around 100 degrees and by birth it approximates 70 degrees. Postnatally, additional slow and slight convergence occurs. From the standpoint of placement of the eyes on the face there is a marked shift from early lateral location to more frontal and central location. At the middle of the second prenatal month the distance between the inner angles of the eyeclefts equals fully three-fourths of the width of the face. During the third month this interocular distance becomes reduced to one-half face width. By birth it is about one-fourth face width, and in early adulthood slightly less than a fourth.

The ears arise at the side of the neck and ascend. The ribs are formed in a nearly horizontal plane and tilt obliquely downward. The ovaries rotate from their original vertical orientation into a transverse position; they also revolve about the uterine tubes and come to rest caudal to them. The nipples move caudad during the early fetal months, register a slight reversal of direction in later fetal life, and change back to caudal migration during postnatal life. In general, they overlie the region of the second ribs in young fetuses and the fourth or fifth ribs in adulthood.

During the last two prenatal months the testes pass out of the abdomen and descend through the pelvic cavity down into the scrotum. In the neonate the intestines and bladder and uterus are located almost entirely within the abdomen; during early childhood they move down into the pelvis. The floor of the maxillary sinus lies above that of the nasal cavity at birth and gradually descends during childhood. The larynx moves progressively caudad throughout postnatal life; the cricoid cartilage approximates the top of the fourth cervical vertebra in the newborn, the bottom of this vertebra at 1 year, the top of the fifth cervical vertebra at 6 years, the bottom of this vertebra in early adulthood, the level of the seventh cervical vertebra in late adulthood, and the level of the second or third thoracic vertebra in late senility. The trachea and lungs show a concomitant downward shifting, gradual during the childhood and adult years and more rapid in old age.

The eruption of teeth into the oral cavity "is but the manifest phase of a process of movement which begins long before."[27] With particular reference

to the upper jaw, at the middle of the prenatal period the teeth (tooth buds) lie packed together in two clusters up near the floor of the orbits. During age intervals of varying duration staggered over the succeeding twenty-five years, each travels "to a position an inch and a half or more below."[28] The corresponding tooth migrations in the lower jaw are, of course, upward. In addition to these long paths of movement from deep within the jaws to the occlusal plane, the teeth make other antecedent, concurrent, and subsequent movements. The precursors of the tooth buds travel from the external surfaces of the maxillary and mandibular processes into their interiors. There is tooth rotation—in fact, "failure to complete the rotation in the ventral plane is commonly responsible for impaction of the last molar."[27] Among the posteruptive changes in tooth position are movements buccally (accompanying increases in the span of the dental arches) and anteroposterior movements.[29]

Other Changes.—A number of structures undergo changes in pigmentation with age. The iris of the eye is characteristically blue or violet in the neonate and, as a result of progressive density of pigment granules, darkens during childhood. In senility there is an increase in the ratio of pigment-free cells to cells carrying pigment, and eye color becomes lighter. The hair of the head shows darkening over the childhood years, followed by graying in adulthood. Except for a difference in timing, the facial hair in males follows a similar cycle of first increase and then decrease in pigmentation. There is darkening with age in skin color, both for whites and Negroes. Negro infants show rapid increase in skin pigmentation during the first two months after birth and much slower increase throughout the childhood years and into adulthood. In white peoples continuing pigmentation of the skin is readily observed in the period between middle age and senility. With advanced age the muscle fibers of the heart are known to become more pigmented and turn a dark brown.

There are changes with age in the texture of various structures. Subcutaneous adipose tissue is less resistant to pressure in the young infant than in the older child. Muscles are more readily compressed or indented in early childhood than during adolescence. Bones are more easily fractured in senility than at younger ages. During childhood there is marked reduction in the water content of most tissues, e.g., skeletal and adipose tissues. Over the adult years there is decrease in organic matter for some structures (e.g., bones) and increase in calcium and iron deposits for other structures (e.g., kidneys). In common with changes in pigmentation, changes in texture have not as yet been as extensively explored as changes in size, shape, position, and complexity. Perhaps the most frequently found change in texture to date is that of decreasing elasticity during adult life. This is illustrated in lessening suppleness of the arteries, in increasing brittleness of the bones, and in declining resiliency of the heart valves.

Finally, attention is directed to the fact that the proposed definition of physical growth encompasses physiologic as well as anatomic changes. While it is not within the scope of this paper to match the above ordering and grouping of present knowledge on anatomic changes with a systematization of physiologic changes, some cognizance should be taken of the breadth and variety of these changes. There are changes with age in such physiologic functions as respiration, circulation, digestion, endocrine output, various operations of the nervous and reproductive systems, visual acuity, muscular strength, and capacity to do work. The heart rate is approximately constant throughout

fetal life, decreases rapidly during infancy, and continues to decline at a slowing rate during childhood and adolescence; it fluctuates readily in early infancy but soon becomes more regular. Heat production and heat loss are higher in infancy than in later childhood; body temperature shows no appreciable change. Systolic blood pressure rises rapidly during infancy and at a slower, fairly constant rate throughout childhood and adolescence. Rate of respiration falls markedly, and becomes more regular, over the first postnatal year; during childhood there is much slower decline and in adolescence only slight further reduction. Over the childhood years there is increase in cardiac output, in creatine excretion, and in the frequency of certain rhythmic electrical activities which accompany cortical metabolism.[30] From early childhood on through adolescence there is decrease in basal metabolism and increase in strength and endurance. During adolescence total respiratory volume increases and the menstrual cycle becomes more regular.

SUMMARY

The foregoing treatment follows a tripartite schema. The first part examines the terms physical growth, physical development, and physical maturation. Attention is given to definitions of each term and to the way the terms are used in research. After displaying materials of both types, the view is taken that the three terms are appropriately regarded as equal in scope and fully interchangeable. Physical growth—the shortest of the three terms—is chosen for use throughout the remainder of the paper.

The second part presents a proposed definition. Physical growth is defined as "the sequence of somatic modification which a biologic organism undergoes during its ontogenetic life history" or, alternatively, as "the entire series of anatomic and physiologic changes taking place between the beginning of prenatal life and the close of senility."

The third part affords a working classification of the anatomic changes currently known to occur during human ontogeny. The classification is indicated to be empirically derived and subject to later extension or revision. As presented and illustrated, it groups the series of known modifications in body structure into changes in complexity, changes in size, changes in shape, changes in position, changes in pigmentation, and changes in texture.

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ORTHODONTIC DIAGNOSTIC PROCEDURES

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WHENEVER a program chairman, regardless of the occasion, falls upon evil days, he reaches deep into the sack of oblivion, seizes an obscure and inarticulate character, hauls him out into the light, hands him a hot potato, and then with a demoniacal leer of satisfaction washes his hands of the whole affair. This explains my presence before you and also explains my subject, which is, Orthodontic Diagnostic Procedures," but which should be, "Let Us Think and Be Sensible." It does not, however, explain why this is a written presentation. As some of you may recall, to your intense regret, I have appeared before you at various intervals and always have spoken without benefit of writing, using only slides or movies as a source of continuity. This is a written paper for two reasons. One reason is that the Program Chairman insisted upon it and the other is that some of the forthcoming statements will be

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very unpleasant and it is only fair to have them in writing so that anyone who might take exception to them will have the opportunity to study them at leisure, in case they are important enough to warrant such scrutiny.

In assigning this subject to me, the Program Chairman suggested that it be kept strictly noncontroversial. Now he should know me better than that. In the past few years I have been excoriated as a reactionary; my treatment methods have been condemned as countrified; my approach to subjects orthodontic has been denounced as confusing, and my insistence upon sequential diagnosis has been dismissed as inconsequential. No one, however, has made the accusation that I was guilty of evasion or dissembling. This will be no exception. It will be controversial, purposely so, and furthermore, it is time that we, as an organization, acknowledge the value of controversy and discussion as a means of progress.

This society is or purports to be a scientific group intent upon furthering the knowledge and understanding of the many problems which constantly afflict us, and yet, almost without exception, we sit somnolently by and allow ourselves to accept, without comment or discussion, statements made before us which should, in the interest of all concerned, be challenged so that proof or disproof may be forthcoming. For my part, should I inadvertently say something important in this paper, it would please me to have it taken up as an issue and I will argue it to the best of my limited ability as long as the discussion remains upon the subject and does not deteriorate into personal fisticuffs. So much for the prelude.

No subject could be chosen which would open the doors wider for the entrance of dissenting opinion than does the one assigned to me this day. This is quite obvious when one considers that we, as orthodontists, have absolutely no common ground upon which a group of us can meet for an intelligent discussion of orthodontic diagnosis, let alone a discussion of the procedures which should be followed. In a group of twenty-five orthodontists it is possible to find almost as many different conceptions of what constitutes an orthodontic diagnosis. That this is true is an inescapable fact as illustrated by the following. At one time I was associated with the graduate division of a college of dentistry here in the city. Amongst my duties was the task of assigning, reviewing, accepting or rejecting the theses submitted for accrediting. One of the students,* dissatisfied with the confusion existing locally in the realm of diagnosis, decided to write his thesis upon this subject. In pursuit of data for his conclusions he assiduously read all the modern textbooks on the subject and, in addition, and this is noteworthy, he wrote to twenty-five representative orthodontists in various parts of the United States requesting information on the methods each employed in making his diagnosis. The results were disturbingly surprising to him. Some ignored the request, so it is safe to assume that these men had no plan. Some replied at length explaining that they had no particular method. Others sent most comprehensive accounts of their painstaking methods and one very honestly replied that his diagnosis was formulated and entered upon the card which he enclosed. The card was small, having labeled lines for the patient's name, address, phone number, and the fee charged. These twenty-five orthodontists ran the gamut from the sublime to the ridiculous and nowhere was there an indication of definite agreement. Why does this situation exist? Why is it so universal even among men who are earnestly

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trying to better themselves, to better the situation, and to better the profession? That is what I shall try to reveal, and in doing so it will be necessary to hold a mirror up to our own activities so that we can see what is going on. It will not be pleasant, and I realize that much wrath may descend upon me. If that is a penalty for speaking forthrightly and honestly, then so be it. Perhaps the end product will be worth the effort.

In order to give basis to the observation that some of this confusion may be eliminated, it might be well to analyze the subject in its literal meaning:

Orthodontic diagnosis is the act of determining in what way and to what extent the denture and related structures of a given case differ from the norm for that particular individual. It is divided into three components: (1) Morphologic, which includes orientation; (2) etiological, which is self-explanatory; and (3) functional. Here is where my conception varies from the conventional. In my opinion the prognosis of the case cannot be divorced from the diagnosis. This statement I shall attempt to confirm later. Nor can the all-important treatment planning be left out of consideration at this point. All of these topics are grouped under what I would prefer to call a five-point orthodontic survey. The term diagnosis is too limited to be satisfactory but is too universally accepted to be replaced.

The word procedure is defined as a "method of proceeding in a process or course of action; order or system of performing, operating, or conducting."

A superficial consideration of the problem presented in the two preceding paragraphs would tend to convince one that the solution to all of our difficulties should be very simple. There are no apparent ambiguities present. The words are simple and easily understood, yet right there in those two paragraphs lies the source of our confusion and dismay. Note well that under the definition of diagnosis is found the simple term "individual norm" and under the definition of procedure are found the words, "system," "method," and "process." There are the jokers and they will be considered at length. First, the words method and system are inoffensive little bisyllabic words, but how many of us can honestly claim that we follow any method or system. Too few can answer that affirmatively. And *why* can't we? The answer to that question is one of the paramount objectives of this paper. Briefly, we do not follow a systematic routine for one or all of three reasons: (1) we are too indifferent to its importance, (2) we have found no satisfactory method, or (3) *we have been convinced that such a course is unnecessary, reactionary, or amateurish.* Of course, a systematic approach to the problem is valueless if the information at hand is inadequate, incorrect, or purposely distorted. It is equally useless if all cases are going to be treated in the same way, regardless of the diagnostic findings. Under such circumstances it makes no difference what approach is employed.

To dispose of the problems contained in the simple phrase, "individual norm" is not so easy and must of necessity entail a bit of consideration, to say nothing of many, many words. So much the worse for you listeners, if any. For a magnificent discourse on the "norm concept" I refer you to the work of Paul W. Simon, entitled, "The Fundamental Principles of a Systematic Diagnosis of Dental Anomalies." He has done it much better than I could, so there is no use in my attempting it. This much I will say, emphatically, it matters not a whit whether the methods of Simon are accepted in toto, in part, or rejected entirely, his discourse on the norm concept is invaluable in planning a comprehensive, logical, and sequential method of procedure. Whatever criti-

cisms may be aimed at Simon's conclusions, no one can say truthfully that his procedure is other than orderly. For my part, I find his methods useful and I employ them within their *rightful* limitations. Whether anyone else does or does not is of no matter for the present, providing that those, who do not, have a reasonable, honest, and justifiable objection to them and have not denounced them merely because the facts revealed by pursuing these methods bring to light many insufficiencies of technique which they would rather not discover.

The basic factor in all orthodontic diagnosis, and we agree upon that as closely as we agree upon anything, is the morphologic factor; and remember that in that consideration there must be a conspicuous place reserved for orientation. In strict truth the term morphology does not include orientation, but I have taken the liberty of placing it there because it is equally as important at all times and, in many instances, assumes the role of major importance. By morphology is meant the shape of the denture, and in a diagnostic consideration it must be assumed that the form of the case in point is comparable to some accepted or nebulous norm which the investigator has in mind. Else, how would the determination of an anomaly be possible? How then does this norm take form and substance? There are many accepted ways, among which are the Index of Pont, the Hawley-Bonwill triangle and the many modifications of these plans. Then too, there are the squint and guess methods employed only too liberally. In any event there must be some preconceived idea in mind before the diagnosis or comparison may be made. It does not suffice to look at the teeth and discover that for some reason or other they do not fit into the arch and something must be done about it. That already has been decided by the patient or the patient's parents or the case would not have presented itself. Granting that the Index of Pont is the accepted means employed, that does not suffice either. It does give a clue to the degree of contraction and that is the extent of its usefulness. It does not give any information on the sagittal, or vertical type and degree of deformity. Nor does it, and I ask you to observe this closely, give adequate information as to what caused the deformity and whether it *can* be corrected completely or partly. That is another phase of the diagnosis. Yet how often does the orthodontist rely solely upon expansion to accomplish his ends.

To return to the orientation of the denture and its degree of divergence from the norm: some form of orientation must be employed. It can be Simon's gnathostatic method, or the Broadbent-Higley teleroentgenographic method of cephalometric orientation employing extraoral landmarks. On the other hand, the various intraoral methods can be employed if found satisfactory. There is the *so-called* keyridge method employing the *jugal buttress of Klaatsch*, or the vertical alignment theory of the lower anterior teeth. It does not make any difference, if the individual investigator can make it work and has faith in it. Personally, it appears to me that the extraoral methods have the advantage in this problem of orientation and I have faith in them. By faith I mean not a blind, unreasoning, prejudicial faith which compels a man to follow to the letter the precepts prescribed and to condemn all else, but a calm reasoning faith which prompts him to investigate the truth of the possibilities, to employ the method within its rightful limits, and to accept its verdict upon his *completed* cases as well as upon his contemplated ones. Please note that comment: To accept the verdict upon his *completed* cases as well as upon his contemplated cases. This statement is not made in jest.

In formulating a diagnosis, it is necessary to investigate the impairment of function existing within the areas under investigation. That, in the case of the denture, is difficult to accomplish because, obviously, we have no way of measuring function in order to establish a norm for comparison. However, impairment of function must be admitted and provision made to restore it to an acceptable degree of efficiency. As far as a diagnostic measurement is concerned, it appears useless except in those rare instances where dental function is so limited as to warrant orthodontic interference in cases which, under *different circumstances,* would be labeled treatment contraindicated. To illustrate: I can recall a case where dental function was impaired to such an extent that orthodontic treatment was employed even though the loss of all the natural teeth was a foregone conclusion. Nor could the case be called a failure as these drastic means made it possible for the girl in question to wear acceptable dentures. She was a congenital syphilitic with the characteristic dental lesions, aggravated by a total maxillary contraction which in closure was completely encompassed by the mandibular denture. Suffice to say, she was treated medically prior to orthodontic treatment until she presented a negative Wassermann.

There are other areas, in addition to the denture, where the type and relative degree of impaired function must be observed and identified. This becomes inextricably entangled with etiology and it is not my purpose to speak at length upon this topic. Nevertheless, in surveying the divergence from normal, thought must be given to the areas of impaired function which are concerned with the case at hand and, also, to determining which areas are the result and which are contributing causes of the dentofacial anomaly. Frequently the circle of cause and effect is complete and differentiation is impossible. In some instances, however, one may be distinguished from the other to the ultimate good of the result.

What causes so much of our confusion in regard to the norm toward which we are striving? The answer to this question is a rather bitter one and I hope that it will be accepted as a helpful one, not merely as a barbed harpoon. Much of our confusion results from plain unadulterated lethargy. Lethargy which prompts us to accept the statements of anyone who chooses to make them without looking beyond the exhilaration of the moment. Particularly is this true if the statements made are of such a nature that they agree with what we wish to believe, or save us some of the tedium attendant upon our daily tasks, or convince us that some of the things we do are right or unavoidable. We are an indolent, prejudicial lot, quick to condemn that which we do not want to believe and equally as quick to accept that which we do. Not only do we apply that policy to the conclusions which are offered us but we also apply it to the men who offer them. If a man is on the blacklist as belonging to another tong, his works, regardless of their merit, are frowned upon and automatically discredited. On the other hand, if he is a good fellow and living in our alley we are apt to accept anything which he may say. None of you will deny that and some of you will apply that same reasoning to remarks I am making here today.

In the first part of this paper I made a plea for discussion and comment as a progressive way of learning what is substantial and what is ethereal, what is true and what is false. I make the same pleas now, because I am going to discuss some of the things said before this group and others, at various times. What Hays Nance will do to me when he takes the floor is in the realm of con-

jecture and no doubt some of it will fall upon me like a bomb. If so, that is good, because I know, and all of you know, that what Hays says will be prompted by what he thinks, not by what he feels toward me personally. I hope you will grant the same purity of motive to my comments.

I said that "a man must have faith in the method of investigation which he employs." The degree of faith was qualified at length so will not be repeated. However, you were admonished to note the statement that "he must have the same faith in the verdict applied to his finished cases as to his contemplated ones." Is it fair to accept the information regarding orientation, derived by a method employed for diagnosis, and then deny it as applied to the finished result? You cannot admit that it is. But such tactics are employed, and I know it does nothing to diminish the confusion in the minds of all of us. This is not a wild, unjustified statement but an observation of my own. I have heard repeated over and over again, before this group and others like it, a long, impassioned, and illustrated speech extolling the virtues of a certain intraoral method of sagittal orientation of the denture. I have heard this same man, having employed a gimcrack appliance which violated every law of elementary physics, make the statement that he had moved all the maxillary teeth posteriorly and reply to a series of questions as follows: (These were my own questions, incidentally, asked in person.) Question: "How do you know you moved the maxillary posteriors posteriorly?" Answer: "I do not know. I think I did but there is no way to measure such movement." Question: "Why not employ the key-ridge, intraoral method which you advocate?" Answer: "Because it is too inaccurate to prove anything." I ask you is that a scientific, unbiased attitude? Why does a man advocate something which he himself does not believe, or if he does believe it, is willing to deny its truth in order to foster interest in a so-called method of treatment? Does that reduce confusion? Let us have honesty upon the part of those who take it upon themselves to direct the destiny of orthodontic endeavor. That *will* reduce confusion.

Not long ago, before this meeting, the statement was made that "Orthodontics is in danger of being set back twenty-five years by a certain man who has attained much prominence by advising the removal of first bicuspids in orthodontic treatment." Orthodontics is not set back twenty-five years by anybody who makes a bold statement, sets out to prove on living human beings that he obtains results by employing the *same* means he advocates, and can show evidence of his success. I do not mean by that that we must all rush out and remove premolars from everybody who presents himself with a malocclusion, far from it! But I do mean that we should consider seriously the results which have been produced by such measures. There are definite, incontrovertible indications for the removal of first premolars. There are equally definite contraindications. Let these indications and contraindications be acknowledged and much thought be given to the in-between cases where doubt exists.

Also heard the same evening was a statement to the effect that "there are rare cases where bicuspids should be removed when the teeth are too big." What determines if the teeth are too big, is it the size of the teeth or the size of the jaws, plus the possibility of growth within the inherent growth pattern of the child? You answer that for yourselves, and don't be too quick to attack the plan unless you can adequately answer it. One thing I know, you can't

answer it without accumulating all the available data, and I mean all that can be accumulated by all available means; and then you sit down and study this information before coming to any conclusion. I make the following statement unequivocably and with no implied apology to anybody. The man does not live who is genius enough to walk up to a child sitting in the dental chair and, with no preliminaries except a cursory ocular examination of the mouth, diagnose the case correctly and outline a satisfactory treatment plan. Nor is any man clairvoyant enough to accomplish this feat by examining a set of denture casts, disassociated from the rest of the organism. Yet I have seen these policies put into practice dozens of times under the guise of education, with results which might be expected.

To journey quickly to the other end of the scale in my discussion of faith in a diagnostic method, I have seen the fanatical person accept, not the spirit of the plan, but the letter in such fervid adherence that he would make his orientation investigations, accept them as the law of the prophets, and follow through to the destruction of the teeth, the face, and the investing tissues. I have seen this attitude encouraged in cases where the orientation conclusions were obviously false, and yet the same man who denied his findings on his own case held firmly to them on another man's case to such an extent that the resulting face was a grotesquerie and the teeth were not even in the potential mouth, let alone the actual one. It is fine to have one pair of shoes to wear when you are out and another more comfortable one to wear at home, but it is nothing less than deception to have two policies of practice—one to prate about and one to employ. That is true for treatment as well. If a man advocates gentle forces in open meeting, he should employ them in his practice, and not whisper sub rosa and intimately, that it is "all right to talk about tiny forces in meeting but if you want to move the teeth you have to bear down." Is that confusing or does that type of thing clarify the air for all of us? Can a statement be a fact on Friday night and a falacy throughout the rest of the week? Let us think and be sensible.

The following statement was made to a small group of orthodontists at dinner one night preceding our meeting, by one of our members. "I do not find it necessary to put auxiliary springs on a lingual arch wire. All I do is place the lingual body wire near the teeth and they move away from it, because Nature abhors the proximity of a foreign object." Is that true, do you believe it? Of course not, but somebody might have, and then spent anxious months waiting for the miracle. I do not say the teeth did not move away from the wire, but I do say, based on observation, that if they did, it was a natural growth impulse and the body wire had nothing to do with it. (Occasionally one of my cases gets better, too, in spite of me.) Why do we deceive ourselves so flagrantly?

Let us consider as briefly as is commensurate with reasonable coverage the effect of appliance technique upon the diagnosis. Immediately there are protests that appliances have nothing to do with diagnosis. In the strict sense of the word they do not. In the daily use of appliances they very definitely do, and I shall attempt to make that statement obvious by the employment of logic, plus honest introspection on the part of each of you. If I am not successful, denounce me, or just forget all about it.

To illustrate: If a man uses a free ligature appliance in conjunction with large arch wires, it is, to all practical purposes, impossible or impractical for

him to accomplish minor rotations. Is he then going to consider these minor rotations in his diagnosis when he knows he cannot accomplish their correction? It is doubtful. On the other hand, if he employs a banded precision appliance in the use of which minor rotations can be easily corrected, he will consider them. This is cited merely as an example. After having treated a few hundred, or a few dozen cases with one appliance or another, a man learns what he can and cannot accomplish. Then he subconsciously accepts as the norm for any given case the best that he can do. That is natural. If he did not, he would of necessity have to admit that his failure percentage was high or change his method of treatment to a better one. This is undeniable. You and I have seen cases without number which one man considered successfully finished and another man would consider just about ripe for treatment. What causes this difference of opinion? It is caused by a different conception of what constitutes the normal. Many of us have seen a clinic composed of 100 cases which is shown in advocacy of the labiolingual technique. To those of us who use attachment bands in varying degrees of multiplicity, these cases appear as though it were time to band them and go to work. To the men who did them the results must appear excellent else they would not exhibit them so enthusiastically. Either these observations prove my point or they do not. In either event enough time has been spent on that.

Nor are we in this area immune to basing our norm concept on the results we obtain. It is a beautiful philosophy if you can convince yourself of its truth, and if you can, more power to you. Others will envy your peace of mind. However, having convinced yourself, it is not conducive to good orthodontics to spread the word around as scientifically true that such results are correct and desirable.

If, in the practice of his profession as he sees fit to do it, a man succeeds in maintaining or creating double protractions and learns to like them, that is no reason why such conditions should be accepted by the rest of us as anatomically correct. It must be clearly understood that the term double protraction refers to an inferior-superior dental or dentoalveolar protraction, not a bimaxillary protraction. These two conditions are not similar. Nor does it convince my stubborn, reactionary, and countrified mind to disinter a horse, amputate his skull, and use it to illustrate how the incisor teeth incline sharply forward from the apical base. Granting that the horse has anteriorly inclined teeth and that they are healthy; the horse is so constructed that he can pick his food off the ground and cut closely to the surface of the earth. That means nothing to us. If it did, the rhinologists would be busy amputating noses and a person with a chin would be a curiosity. We have nothing in common with the horse except that both are warm-blooded, mammalian vertebrates. Such a flimsy analogy is of no value. Nor is my bewildered mind impressed by resorting to chummy little talks about the birds and the bees, explaining that, "A little child is like a young bird who is all mouth; as it grows the rest of the face will grow to fit the mouth." Perhaps the horse is the better analogy of the two. At least the horse does have teeth. Let's be honest with each other. That is the best way to eliminate confusion.

Not so long ago many of us were convinced that in the correction of Class II cases, involving a superior dental or dentoalveolar protraction, it was a simple matter of a few months and very little trouble to move all the superior buccal teeth posteriorly. We had been told repeatedly that "there always is plenty of room back there, as much as you want." So we tried it. In some

cases it worked, in others it did not. I have cases where it did, as measured cephalometrically, and I can show cases where I thought it did and it did not. Then there are plenty of them where I couldn't even fool myself because quite obviously the lower denture was moving forward. What was wrong? Is there plenty of space back there? There is not. I do not have seven or eight hundred denuded alien skulls, whose original facial contours are unknown but racially do not in the average resemble us Americans, to prove or disprove my point, but I do have experience involving the treatment or observation of many hundreds of children and I am convinced that there is not always plenty of room behind the maxillary posterior teeth. Perhaps I can offer sufficient evidence to prove that to you and thus save some of you weary and unsuccesful efforts. Dr. Oppenheim, with whom I have enjoyed some most profitable arguments, always insists that an opinion is not worth anything per se and must be proved. Proof is an intangible when dealing with intangibles, but, if enough evidence is offered, one is inclined to believe. Again, let me repeat, if it does not satisfy your demands, ignore it. First of all, let me state again that in my own experience some of the cases lent themselves to posterior movement of the maxillary buccal sections and some did not. Obviously there are many complex factors to be considered there despite the fact that the same methods were employed in all cases, but it does lend credence to the supposition that the posterior area is crowded. Then too, I have observed, as undoubtedly have many of you, that when an unyielding attitude is assumed in this procedure and the attempt to move the sections posteriorly, or even units posteriorly, is stubbornly continued, the unconfined maxillary second molar is squeezed out to the buccal. Would this tooth take the hard way through the buccal cortical plate if there were room for it to move directly back through responsive cancellous bone? It is doubtful. Then too, consider for a moment why the segments have moved forward, granting that they did. Was it because of some posture or pillowing habit or was it because there was insufficient room for the encroaching second and third molars to assume their correct positions? As to the effect of pillowing or other posture habits I am not prepared to say. Most of us are convinced that these pressure habits have a detrimental effect upon normal maxillary growth and orientation, but again the proof is nebulous. However, to digress for a moment, it is suggested that you watch for the work of Dr. Vern Fluhrer, whose investigative turn of mind led him to arrange an automatic camera which takes periodic exposures at time intervals during the night. This little contrivance, placed on the night stand of his patients, records a series of posture pictures. When completed, his investigation will have a marked effect upon our conclusions regarding pillowing habits.

To return to the problem of the superior retromolar area. Many of the second molars in the cases of superior protraction have erupted crowns, the occlusal surfaces of which incline sharply upward and backward and the roots incline sharply upward and forward. This is due, in my opinion, to lack of posterior space for the root to move back and allow the tooth to assume an upright position. Not conclusive proof by any means and yet sufficiently significant to be worthy of thought. This next one is a fact. A local exodontist, of indisputable integrity, was asked, without any explanation, for a tabulation of the reasons why the maxillary third molars which he had extracted had to be removed and what position they occupied. This, mind you, is in adults, whose facial growth and anterior development, including tooth migration, were

complete. How many of them were superior protractions with all the buccal teeth already forward is not known. However, it is significant to note that he has removed approximately 28,000 maxillary third molars. Of this number nearly 9,000 were impacted with no room for eruption, another 9,000 had erupted buccally, the remainder were removed because of caries or partial eruption. The evidence is accumulating. The remaining 10,000 appeared to have normal position, but to quote the exodontist, "the things were always decayed." This carious condition could well be the result of faulty formation of the crown due to the constricted area in which it was compelled to develop. That is not made as a statement, merely as a supposition, and one which you may accept or dismiss. You may well ask yourselves if there is plenty of room for posterior movement of maxillary buccal segments. Denuded, dried, alien, or animal skulls do not provide the answer.

Of late we have heard much of osteoporosis. In a vague and general way we have become convinced that it has something to with orthodontics. What is osteoporosis? Literally it means that the bone is porous. All bone is porous, so nothing is settled by that admission. Why then should we be concerned? Obviously because there is a normal, characteristic architectural porosity which is healthy and beneficial to us in our work. If this porosity increases beyond normal bounds, it impairs or renders orthodontic procedures impossible or even destructive. Just to talk about osteoporosis means nothing unless the condition is one which is recognizable, demonstrable, and something is done about it. Before it is recognizable, some thought must be given to the type of porosity manifested. Is it the typical, accepted picture of von Recklinghausen's disease, or is it merely an illusion established by diminutive structures with closely approximating cortical plates? Between these two extremes, one most serious in its systemic implication, and the other serious only in that it must be recognized by the orthodontist and his conduct governed accordingly, there are many different indications of bone deficiencies revealed by an abnormal radiolucency or radiopacity of the alveolar areas. Among them are the familiar pictures of dystrophy, the areas of osteodystrophia cystica and fibrosa as recognized by Becks. Are these areas of bone dystrophy the result of dysendocrinism, avitaminosis, simpler metabolic insufficiencies, or merely inadequate food ingestion? These questions must be answered, and many more, before any benefits accrue in the consideration of osteoporosis. In case of endocrine dysfunction, the orthodontist is not, in my opinion, capable of assuming the responsibility of the endocrine diagnosis and treatment by himself. Endocrinologists themselves admit much puzzlement in their field and we have made no study of the subject comparable to theirs. I do believe, however, that when suspicion is directed toward endocrine dysfunction by the oral radiographs, immediate steps should be taken to confirm or deny these suspicions by the simpler means at our command. We are, or should be, familiar with the over-all superficial picture of dysendocrinism, and some of these indications are surprisingly accurate in the more active cases. Where they are present or where the suspicion persists, it is a simple matter to obtain profile skull and carpal radiographs to confirm or deny the suspected condition. Then it is time to call in the services of the endocrinologist in whom you have confidence. It is not meant by this that a shotgun injection of thyroid or pituitrin is indicated, as so many of our pediatricians seem to think will satisfy us. In my own work I have been fortunate enough to detect many endocrinopathic conditions in children by simple means and so far not one of them has failed to reveal glandular disturbances

when competently surveyed by an able physician. It is most gratifying to observe the changes, almost metamorphoses, which take place in growing children when glandular imbalances have been corrected. It is true that not all of them respond, but when they do not, orthodontic procedures can be modified to give the most satisfactory results while at the same time minimizing the degree of jeopardy to which the tissues are subjected. This subject of endocrinopathies and their relation to orthodontics has been most successfully and fully investigated by Dr. Sydney Cross and reported in his thesis, submitted for accrediting a few years past. Dr. Cross' work was done as an original investigation and he secured the interest and assistance of Drs. Tager and Wright. Both of whom are recognized endocrinologists. It is suggested that this work be reviewed.

In discussing various treatment methods with an orthodontist some time ago, it was a bit shocking to hear him say, "What takes place under the alveolar mucosa is none of your business." I insist that it is your business, and my business. It is in the alveolar mucosa and the tissues underlying it that our work, for better or worse, is done. The more knowledge we have of what goes on in investing tissues under the stresses to which we subject them, the fewer mistakes and abuses we will inflict upon them. That we abuse these tissues every day is an admitted fact, and none of us is so blind as to believe that the stresses we apply are truly physiologic. Some of us approach this ideal more closely than others but none of us actually accomplish it. That our efforts frequently succeed is due to the gracious faculty which living tissues have for absorbing abuse and then recovering from it. For the sake of convenience I have taken the liberty of applying the term "limits of tissue tolerance" to the varying amounts of change, artificially induced, which a living tissue will tolerate. That there are varying degrees of tolerance or response indices manifested by different tissues and different individuals is a matter of common observation to all of us. In order to amplify further this distinction I have formed the custom of refering to the differing degrees of tissue tolerance as "high tissue tolerance" and "low tissue tolerance." Each one of you has seen superficial evidence of this in his own practice. Superficial tissues with high tolerance will remain healthy, clear-textured, and firm despite the many irritations accompanying appliance placement and tooth movement. Other mouths, whose superficial tissues have a low degree of tolerance, become congested, hyperplastic, flabby, and loosely attached in spite of all efforts made to treat them kindly. There is no reason to suppose that the underlying tissues are any different, and honesty will prompt each one of you to admit that, following tooth movement which was not radical nor extensive, you have observed tissue loss and resorption, not only in the superficial tissues, their immediately underlying bone structures, but also in the roots of the teeth themselves. What causes this in one case and not in another of almost identical type and under comparable conditions of treatment? My explanation is that in one case the tissue tolerance and growth index was high and in the other the tissue tolerance and growth index was low. What determined this is not always known. However, it is known and demonstrable that the basic systemic background, the metabolic efficiency, and the care taken in adapting the demands placed upon the tissue to the limits of its tolerance, are important factors. What takes place under the mucosa, for that matter what takes place all through the organism upon which you are working, is definitely and indisputably your business, and my business. The area in which we are working, whether our

objective be the creation of facial harmony or the simple correction of a minor malocclusion, is not an isolated organ which just happened to be fastened to the body but is an integral part of a living organism. Having accepted the responsibility for the area immediately concerned in our operations, we must acknowledge the responsibilty for the whole organism. The denture and the immediately contiguous areas did not become malformed of their own volition. They are malformed because the parent organism, if that misapplication of relationship may be used for purposes of simplicity, failed in its support and guidance to the dentofacial area when this area was developing. So, the more we know about the whole organism the more we shall know about the tissues underlying the alveolar mucosa, the more we know about them the more we shall know about what effect we have upon them in treatment, the better we will uderstand the possibilities and limitations of mechanical therapy, and the better orthodontists we will be. Gentlemen, what goes on under the alveolar mucosa and what goes on in the whole organism *is* your business.

In case such generalities do not suffice to convince every one of us that in assuming responsibility for orthodontic care of an individual we do by the nature of our efforts assume responsibility for the whole individual, at least in so far as his organic processes concern themselves with our efforts, a bit of emphasis by way of illustration might be apropos. In the interests of brevity, four illustrations will suffice, although the list is endless.

CASE 1.—The first one concerns the efforts of two orthodontists who tried consecutively to bring into position an impacted maxillary canine in a young adult. The total time expended was close to three years and the tooth had not moved from its position to any visible extent. In fact it had not moved at all. Careful scrutiny of the radiograph revealed the reason, and either of these orthodontists should have known before making the attempt that the tooth could not be moved. The reason was simple. The tooth was ankylosed and being ankylosed, had no periodontal membrane. Without any periodontal membrane there is no possibility of osteoclastic-osteoblastic action, and without this action there is no possibility of changing the position of a tooth by growth processes.

CASE 2.—The second case was that of an incarcerated maxillary left first molar. This case, with a full complement of permanent teeth, also had been under treatment for more than a year. In the attempt to cause the incarcerated molar to assume its rightful position in the plane of occlusion the denture had been disrupted until only six teeth, on the right or opposite side of the denture, remained in occlusal contact, the rest of the bite being open. What caused the first molar to resist so heroically all attempts to extrude it? It was in an area of osteonecrosis. Such an area of bone, while comparatively soft, has no living osteophytes, so tooth movement through growth processes is impossible. Obvious, of course, but overlooked by insufficient interest in what goes on under the alveolar mucosa, and an inadequate diagnostic procedure.

CASE 3.—This was a young adult woman who had been under treatment for an interminable time with unsatisfactory results and was at the time of my observation under further treatment, the word being used loosely, by a so-called active retainer—the type of active retainer which is worn only during the night to retract the maxillary anterior teeth so that the extruded lower incisors can batter them forward during the day. When she came under my observation, she had been to a periodontist to see if her teeth could be saved. Some of them were saved, but she lost, as the result of alveolar absorption, all of her maxillary anteriors including the canines, the lower incisors, and one lower canine. Also three first molars were so involved as to be beyond saving. The loss of the maxillary six anteriors could be justified by procuring the skull of a common cow, which is a warm-blooded mammalian vertebrate and has as much in common with the human being as the horse, and using this as evidence to illustrate that maxillary anterior teeth do not belong there anyway. In case this puzzles you, a cow does not have maxillary anterior teeth. Even this subterfuge would not justify the loss of the other teeth, so some investigation is in order. Of course

each one of us can say, "That would never happen to me." But it could and probably will some day unless a definite, logical, and sequential diagnostic and treatment-planning procedure is adopted. This tragedy could have been avoided by employing some such policy in this case. Such a policy would have revealed, as it did belatedly, that the young lady was a diabetic, her basal metabolic rate was a constant—25 to 30 *despite* the fact that she was under daily treatment with extract of thyroid and pituitary. All of this was unknown by the orthodontist who was interested only in what he saw in his casts. To make the error more flagrant, the girl's father was a physician and would have furnished all this information had he been asked or had the orthodontist made any issue of need for such information. You might well say, "What of it, just a few minutes ago you told us of a case where *all* the teeth were removed subsequent to orthodontic treatment and the case considered a success." So I did, but the comparison is not a valid one.

In the case under discussion no good was accomplished nor was the damage done unavoidable. It could have been avoided and the case improved by recognition of a simple principle which is, "Bone which cannot maintain itself because of metabolic insufficiencies cannot increase under mechanical pressure nor will it survive these pressures." That statement will not be found in any textbook because I formulated it myself. To me it is common sense. You are free to accept it or reject it as you wish. My only request is that you think it over before coming to any conclusion. In defense of the statement the following is offered: That it is my own case does not in any way reflect credit upon me as an astute investigator nor as a brilliant orthodontist. I make no claim to either. However, in a case presenting exactly the same systemic background some success was achieved by recognizing the condition prior to treatment and modifying the treatment in accordance with the possibilities of success. Some bone and mucosa were lost around the gingival borders of the teeth, no teeth were lost and the case has held up now for over ten years. The dental function is average, the appearance of the mouth good and the face immeasurably improved. The difference in treatment was most radical. In accepting my axiom regarding the inability of the bone to grow, the treatment was so planned as to cause all tooth movement to remain within the already established limits of bone. Which engenders another axiom of my own, humbly offered for your consideration: "In treatment of adults or children with subpar growth possibilities it is advisable to confine all tooth movement to such as can be accomplished within or toward the already established limits of bone." In so doing, in the case which has just been briefly reviewed, it became necessary to remove four first premolars, limiting tooth movement to the retraction of the anterior twelve teeth, plus minor positioning of the posteriors, all of which could be accomplished and *still remain within the established limits of bone*. The teeth were *not* removed because they were too big as judged by millimetric standards expressed in the various indices, but because they were too far forward, the arches contracted, and there was no hope for the diminutive, deteriorating bone to accommodate itself to them. It makes no difference what the actual size of the teeth is, if the bone cannot accommodate them they are too large. How much better it is to learn and accept these conditions prior to orthodontic intervention than to wait until afterward and then take refuge in them.

CASE 4.—Some of you have heard of this one and seen the slides on it before. Those of you who attended the summer conferences, given at the local dental college, at which I persisted in throwing the ranks into confusion by my rebellious refusal to accept whimsy as factual procedure, will remember it. It would be interesting to see how many of those who have not seen it would recognize the evidence if presented. However, by the time this

paper is finished no one will be in the mood for slides or other time-consuming inpedimenta so none will be employed. The case was a lad who had been under treatment by a conventional appliance, and a very good one it was too, for several years in the attempt to correct a Class II, Division 1 subdivision malocclusion, or what seems to me to be a more satisfactory descriptive nomenclature, a unilateral mandibular retraction. The progress of the case had been uneventful except that the unilateral mandibular retraction or asymmetrical Class II malocclusion did not show any signs of response. It took but a moment to detect, by palpation, that the condyle on the retracted side could be held under suspicion. A lateral jaw radiograph revealed that *no condyle* existed on that side. How then could anyone hope to establish and maintain normal intermaxillary relationship by means of Class II intermaxillary rubbers? It could not be done. The only alternative was to train the lad to develop a unilateral protrusion which could be maintained by the musculature. Such was done with moderate success.

This also is cited, not to discredit anybody but to show how much trouble could have been avoided by the simple inflexible purpose of determining *all* the conditions prevailing in a prospective case before treating it. How many of you take the time to palpate the condyles? Maybe all, maybe none, and the chances are that if you do you will never find another condyle missing. (I have found but one other case, a child with no condyles.) What if you don't? It takes but a minute and the reassurance is of value. In case you have lost sight of my purpose, I still am appealing for and attempting to justify the need of logical, sequential, all-encompassing diagnostic procedure. Once it becomes a habit, it no longer is burdensome.

In citing Case 3, where the statement was made that under certain conditions, success and safety can be assured "only by remaining within or moving the teeth toward the already established limits of bone," I have stepped into a potential hornet's nest. It was done deliberately, not merely for the sake of controversy but acting upon the conviction that a little light shed in the dark corners is better than none. Maybe my efforts will result in no improvement and maybe they will stir up a bit of thought. That is all I hope to accomplish.

Case 4 was an extreme one which anybody could have recognized had he employed ordinary caution. Such cases are not the bewildering ones. It is the great mass of cases that lie between the obviously handicapped ones and those which are equally as obviously in the realm of excellent prognosis which are troublesome. How many such cases do you find? Most of our work lies within that category, and most of the failures, yours and mine, are the result of misinterpreting which is which. Note the inclusion of my own mistakes. I have made many, will make many more, and without doubt some of you men have encountered my mistakes wandering around the country in various states of collapse. That is not the point, and the reason I have chosen the citation of errors as a means of illustration is not to place the onus upon anybody else. It has been done merely for emphasis. The point is that all of us make too many mistakes. Incidentally the reason for the nauseating repetition of the pronoun "I" is that I am talking only for myself and whatever violent repercussions may be attendant upon this paper can be directed to the proper source, which is I. No one else is responsible. Now with that bit of explanation, let us return to the subject and further discussion of the possibilities of bone accommodating the normal complement of teeth, placed in accordance with accepted morphologic norm.

It is necessary, as mentioned previously, to accept some arbitrary norm, else there is nothing upon which to stand in making a differential diagnosis,

morphologically. It also must be assumed in commenting further upon this problem of bone growth that the appliance used to induce this growth is an acceptable one with sufficient versatility to apply the mechanical stimuli in the direction and to the extent required. Most of us use such a mechanism, *but* most of us also place too much faith in it. It is almost characteristic of us orthodontists to assume that we can do what we want to do by virtue of the appliance in use and to assume responsibility only for its actions. Too frequently the changes in the shape of the denture are brought about by mechanical means, and then we shut our eyes to what changes, if any, have been brought about in the supporting and investing tissues. If it has been found upon the completion of treatment that the changes in form and orientation of the coronal portions of the denture have not been reflected in the unseen portions of the teeth, their investing and supporting tissues, it is very consoling to assume that subsequently these changes will be manifested. Meanwhile the case can be kept under retention while the desired changes are awaited. That these changes frequently take place, within limits, both during and subsequent to active treatment is an acknowledged fact and this fact has been demonstrated very capably by various investigators. Unfortunately, however, not enough significance has been attached to such instances where the desired changes in the form, size, and orientation of the bone, in its entirety, do *not* materialize. These cases must of necessity fail, and yet so often the orthodontist shuts his eyes to this fact and finds solace in the self-persuasion that he has done all he could and that from now on it is up to Nature. It must be borne in mind that Nature already has failed once on this particular individual or he would not be in the hands of an orthodontist.

There are certain rules of conduct prescribed for the actions of bone under various conditions, and, in the main, bone lives up to these expectations. It is the exceptions which provide us with our daily disappointments, together with a thoughtless acceptance of some of the rules laid down by former investigators. In attempting to justify an unreasonable degree of tooth movement, in which the teeth are tipped or moved bodily off their supporting base, it is not an uncommon practice to seize upon the almost universally accepted "Wolff's Law." That there is merit in the findings of Wolff is not to be lightly denied, and, if it were, I am not the one to do it as my observations are not based upon microscopic findings. They are based upon clinical observations of treated cases, my own and those of many other orthodontists, where the law failed to work during active treatment and again failed to provide the bone changes required for stability even after eight and ten years of retention. Nor are these observations limited to the use of any one type of appliance. They include all known acceptable types and even many inadequate and antiquated appliances which are periodically disinterred for no known reason.

Before going any further into this phase of the subject, let me ask you a question. How many orthodontists who so glibly refer to Wolff's law know what it is or where it came from? Each man apparently has his own conception of what the so-called law includes, and applies it as he sees fit. Many of you are going to be a bit abashed when you learn what the law actually states, where it came from, and when.

Julius Wolff, born 1836, died 1902, Professor of Orthopaedic Surgery in Berlin, became interested in the changes which occurred in architectural conformation of the bones during the period of their development, and its subsequent adaptation to changes of stress and

strain during the recovery period following trauma or deformity. Morphologists had long recognized the general principle of functional control of growth. It was known, for example, that in the development of the limbs the skeletal tissues changed their axial directions to conform to new lines of stress and strain.

Between 1868 and 1892 Wolff stated his concept and in a lengthy monograph* published in 1892 attempted to establish it as a mathematical truism which could be accurately predetermined. He held that "every change in the form and function of a bone, or of its function alone, is followed by certain definite changes in its internal architecture and equally definite secondary alterations in their external forms, in accordance with mathematical laws." This has since been known as "Wolff's law." Although the principle underlying this observation was not original with him, Wolff tried to demonstrate that these changes were not casual but were produced in accordance with mechanical laws of pressure and tension. He attempted to evolve mathematical formulas to express the nature of these changes, but, as is always the case in complex biologic problems, the variables were too numerous and too complicated to permit of accurate tabulation.†

Much to my dismay, I find nothing in this verbatim quotation which leads me to assume that the bone of the maxilla or mandible will grow and conform itself to needs which are placed upon it by the mechanical positioning of teeth outside the prescribed area of the mandible or maxilla. Mind you, I am not discrediting the findings of Wolff. I am merely calling to your attention that in our anxiety to believe we are doing the right thing under all circumstances, we have been or are prone to read into such laws, comforting statements which do not appear under unbiased scrutiny.

To repeat; no one denies, it would be folly to do so, that bone changes its structure, architecture, and form under functional stress and strain, but to what extent? Accepting the most optimistic interpretation of the law explains our successes and the accompanying or ensuing bone growth and adjustments. However, and this may startle you, it also is possible to explain our relapses by application of the very same law. To quote in part from the paragraph preceding the concise statement of Wolff's law: changes which occurred in the architectural conformation of the bones during the period of their development, and *Its Subsequent Adaptation to Changes of Stress and Strain During the Recovery Period Following Trauma or Deformity.* Could it not mean with equal verity that the bones which we have distorted by our orthodontic interference will change in form and structure so as to return to their previous shape, size, and architecture when the stress and strains are removed? How often have we been the cause of the "trauma and deformity" from which the bone is recovering and changing its form and architecture? Answer that before you feel safe in assuming that any change you make in the placement of teeth is going to be compensated for by a change in the bone in keeping with the newly enforced conditions. Nor does it suffice to dismiss the topic with those comments—comments which appear, on the face of them, to be iconoclastic and disturbing, but none-the-less thought provoking.

How great a part of bone growth is dependent upon function, and by function is meant natural physiologic function, and how much of it is determined by the inherent growth pattern or the genes contributed by our progenitors? Not a small influence is exerted by these unknown factors which determine the existence of every individual.

It must be acknowledged that while each human being presents discernible differences in facial characteristics, resemblances remain and are transmitted

*Wolff, J: Das Gesetz der Transformation der Knochen, Berlin, 1892. (The Law of the Transformation of the Bone.)

†Bick, Edgar M.: Source Book of Orthopaedics, Baltimore, 1937, Williams & Wilkins Co.

from generation to generation. In every tabulation of etiological factors governing malocclusion will be found an admission that heredity plays a substantial part. Different families present recognizable characteristics as to height, form, hair or lack of it, facial contour, and skeletal development. Conversely, members of the same family will show gross differences in skeletal development although following the same pattern. Each of us is an individual endowed with at least two growth factors: growth in anticipation of function, and growth in response to function. Do not overlook that statement, "growth in anticipation of function." It is vitally important and to my mind plays a tremendous part in the ultimate size of our faces and jaws. Nor can that statement be denied by replying that the major part of our skeletal growth takes place after function has been established. True, it does, but remember that the inherent growth and growth-inhibiting factors do not cease their activities because of functional influences. Both of these factors continue to exert their influence as long as growth takes place in the individual. Which is the more powerful remains open to discussion.

There is one point, however, which is not open to discussion. Tooth growth is stimulated solely by the inherent factors and is completed in anticipation of function. Teeth are nonfunctional tissues, whose size and shape are in no way influenced by the use to which they are put. Not only that, but the teeth complete their growth, as far as space needed to accommodate them, long before the maximum growth has taken place in the rest of the organism. Tooth formation, except for the roots, is complete even before the major implements of maturation reach their maximum activity. What has that got to do with bone growth and Wolff's law? Just this. There are inherent factors in the growing body which determine within absolute limits what the size and shape of certain organs will be. It so happens that there is incontrovertible evidence in the case of teeth.

Also, as far as available, accredited sources of information reveal there has been no change in the number of teeth in the human skull and no consistent reduction in the size of the teeth during the evolutionary progress. The skull of the Neanderthal man, the Heidelberg man, and the Pekin man, which to my knowledge are the most remote of our ancestors, contain the same number of teeth in the same relative sizes as are found in the heads of human beings today. Yet, the prognathic faces of these prehistoric men in no way resemble the faces which we accept today as being normal or even pleasing in appearance. H. Wingate Todd, in describing the Heidelberg man, says in part, "He was large jawed and beetle browed . . . . . The teeth though retaining many of the ancestral features are typically human in form and no larger than those to be found in many modern Australian skulls." Also they are the same in number. This is in reference to the oldest recognized form of man who lived by his strength and inhabited the earth during the Glacial period, an estimated 250,000 to 1,000,000 years ago.

Again quoting from Wingate Todd, "All human races of today are obviously different from the Heidelberg man in possessing greater tongue space and SMALLER jaws, though not necessarily smaller teeth. They are unlike the Neanderthal man in the possession of a chin."

Also referring to the Heidelberg Man, this statement appears: "In the great vertical depth of the body, the breadth of the ramus, the form of the

symphysial region and the ABSENCE OF AN APE SHELF . . . " This ape shelf obviously refers to the simian ridge found occasionally on untreated modern man and all too often on orthodontically treated children.

It appears that 250,000 to 1,000,000 years ago Nature through the process of evolution eliminated an undesirable vestigial anatomic characteristic which we as orthodontists are now encouraged to recreate.

The inherent growth factors regulating the size and number of teeth have not altered, but no one can deny that with the exception of the Negroid type, the classical anthropoidal prognathous face has been lost in the evolution of man. Maybe that is good and maybe it is not. I merely suggest that you think it over before trying, in all cases, to crowd thirty-two or even twenty-eight teeth into the mouths of contemporary human beings.

In the consideration of the growth of bone it is more difficult to prove the influence of these inherent growth factors. Nevertheless, consider this report if you will: It is known as "Keck's case" and, although an isolated instance, it gives credence to the assumption that too much faith is placed in the application of the rules governing functional factors in growth.

Keck's case is the report of a child born deformed. The deformity was of such a nature that the fibula of one leg was laid down outside the normal integument in such a manner that it had absolutely *no* muscles attached to it, yet it grew, keeping pace with the normal fibula in the other leg and had every evidence in the bone surface of the irregularity normally attributed to muscle attachment and stress. How much of the facial bone growth is functional and how much inherent? Surely that is food for thought and a possible clue to the cause of some of our failures.

All of us have had the experience, in Class II, Division 2 cases, better described as a mandibular retraction in conjunction with a superior anterior dental retraction and abstraction, that when in the course of treatment the maxillary incisors were intruded and inclined toward the vertical, the mandibular retraction became corrected almost before the Class II intermaxillary elastics were utilized. How did this phenomenal correction take place? It could *not* have been in response to function because the abnormal function during the malocclusion was impotent to do it and the correction of the retraction was manifested *before* normal function could be established. What then caused it? In my opinion it was caused, not by any magic of treatment, but merely by removing the inhibiting action of the maxillary incisors and freeing the *inherent growth impulse*. Nor would that have sufficed were the inherent stimulus to normal growth submerged by a malfunctioning organism.

Does it seem sensible to diagnose and outline a case of orthodontic treatment involving interference with the growth processes in bone without exhausting every available source of information? Even that is pitifully little. Maybe a policy such as I am advocating is confusing to consider, although disconcerting would be a better word, because it means to the complacent and indolent that there is much work to be done in orthodontics and that "squint-and-try, do-as-I-say" methods of diagnosis and treatment planning are as impotent and inadequate as some of the "spit and string" appliances which are periodically resurrected from a well-deserved and all too shallow grave.

This could go on interminably and it probably seems as though it already has, so if you are not convinced by now that there is a crying need for an all-encompassing, logical, and sequential diagnostic procedure, you never will be.

In case you ask what should be done about it, if you are guilty of the inadequacies and mistakes called to your attention in this paper, and what man is not guilty of some of them, my reply is this: think before you accept dicta, think before you accept repudiations, think before you accept panaceas, think before you accept short cuts, think before you believe, think before you allow yourself to be led into accepting any comforting solution to your problems. Orthodontics is not a restful profession and there is little comfort to be found in it because we are not too well informed of what we are trying to do or what we actually can do.

I do not believe that orthodontists as a group are "so dumb that you can tell them anything and they will not know the difference." I do not believe that orthodontists are intellectual paupers, groveling at the foot of Olympus and obsequiously grateful for whatever might be tossed to us from those on heights. At least I do not believe we are unless we want to be. I *do* believe that we are too prone to accept what is offered us, and much of it is offered in all sincerity, in whole or in part, and then to misuse it. This tendency is visible every day in our contacts with one another. Just as a man in dire distress will seize anything offering him support, so do we orthodontists who are cognizant of our shortcomings seize everything that might be helpful to make our work easier. Then, having seized upon a pertinent fact or two, we toss it into a jumble of unassorted information and misinformation which we carry around in our heads much as a housekeeper throws this and that into the basement. Then from a mass of unassorted, uncorrelated miscellany, much of which should be discarded, we seize an item or two as we plunge headlong through a hasty diagnosis.

After all this criticism, you may expect that I have a beautiful solution to all of our problems. Well, I have not and I frankly admit it. I do not know all the answers. Nor does anybody else, but I do believe that by following an orderly series of progressions in unison, eventually we will find most of the answers to our vexing problems. At least I can offer a plan which will eliminate much of the confusion attendant upon diagnosis, and open vistas for constructive *thinking* which will reduce the incidence of failure both during and after treatment. It will not take very long because it is a bare outline, the successful application of which will depend upon the thought and careful consideration devoted to its application.

No attempt will be made to give in detail all the recommended points of consideration nor the actual routine of investigation as that would require a day in itself and necessitate the recitation of innumerable histories in order to justify the apparent irrelevancies which are utilized. It will suffice for tonight to lay down general rules of conduct in a comprehensive diagnostic procedure and from that nucleus each man may readily develop a formula of his own or call for additional information at some future date.

The First Step.—The most obvious and conventional approach to the actual diagnosis of a denture anomaly is that of determining morphologic and orientation deviations from the norm. That is a simple matter if a method is accepted which is reliable, flexible, comprehensive, and useful. Remember this: To be reliable in determining the pretreatment abnormality the method employed must be granted the same degree of accuracy in determining the post-treatment approximation to the norm as is attributed to it in the pretreatment application. To be flexible, it must not be dependent upon any arbitrary law which cannot be adapated to the variance in face forms. To be comprehensive

it must determine not only the type of the abnormality, but also, and this is important, the degree of the abnormality. To be useful, it must lend itself to daily application without involving undue complexities of operation.

Having determined the type and degree of the denture abnormality, this information must be transferable to the face by means of orientation photographs or radiographs. The isolated denture reproduction is too limited in scope to be reliable as it presents only the immediate area of local deformity whereas our major interest is or should be in the contiguous and adjoining areas, including the *whole* face. The face must be analyzed for its basic form, racial characteristics, skeletal outline, extent of maturation, inherent growth pattern, and the possibilities of adapting the denture not only to the present limitations of growth but also to the potentialities of growth. So far the problem is one of arbitrary norms and mechanics.

The second step is that of a complete radiologic examination of the denture, its investing tissues, the maxilla and mandible in their entirety. If necessary, using a complete intraoral series augmented by the usual extraoral or lateral jaw pictures. A careful interpretation of these films includes all the obvious considerations such as number and location of teeth, root formation, progress of dental maturation, gross measurements of all bone areas visible, etc., *plus* assiduous attention to the details of bone development as to quality and architecture.

The Third Step.—Third, and here is the weakest point of all, the determination of the factors causing or contributing to the malformation. While this is a weak point in diagnosis it is a still weaker point in prognosis and treatment planning. Our information being admittedly inadequate in this field, there is a tendency to ignore the whole subject and resort solely to symptomatic treatment. That is a mistake. The least that can be done is to make a tireless effort to determine the causes, and then, and mark this, redouble those efforts in the attempt to learn if any or all of the etiological factors are still operative. If so, do they lend themselves to corrective measures or must they be tolerated and the situation accepted with the best grace possible. That there are remediable causes and irremediable causes can be simply illustrated by thinking back over your own cases. However, to attempt orthodox orthodontic correction in the face of irremediable causes is as impractical at attempting to wash your hands in a bucket of mud. All the known means can be employed without success. It will take but a moment to give an example of that. It is a personal experience and not a very flattering one. The case in point was my first orthodontic case and I worked upon it for five and one-half years. Briefly, it was a Class II malocclusion characterized by a superior dentoalveolar protraction and complicated by an anterior open-bite. To be just with myself it must be admitted that the denture reproductions were submitted to various other orthodontists for assistance and from nowhere did the solution come until it developed itself. It was from this experience that the term "anesthesia throat" took origin. I coined it for lack of a better one and it describes a throat which is immune to sensation, having no sensory nerve supply. In such cases the individual has no normal impulse to swallow nor will he gag upon touching the uvula, velum palatinum or any of the usual trigger points in the throat. In order to swallow he must seize the tongue between the anterior teeth and by dint of muscular contortions, involving even the face, perform the act of deglutition. What chance has anyone to correct and maintain the correction of

an open-bite under such circumstances, and how simply one can determine this condition by watching closely the efforts of the patient to swallow and then, by means of a mouth mirror, test the swallowing reflexes. Trouble, disappointment, and much unrewarded effort can be avoided by this simple act, as was the case of the missing condyle. And how little time it takes to do these things.

The Fourth Step in the recommended procedure is to record the physical findings in the case of the individual under observation. The extent of this investigation is dependent upon many things, not the least of which is the age. In the formative age, which is the usual age for our patients, it is important to observe the child from all aspects, such as general appearance, medical history, chronic ailments, posture, fatigue index, appetite, growth, development (and incidentally these are two words which are kicked around with little or no knowledge of the difference in meaning), tongue, nose, throat, skin texture, mentality, dexterity, and so on through a long, long list. That the apparently irrelevant characteristics are important will become self-evident if you will mentally review the symptomatology of the nutritional and endocrinologic diseases and insufficiencies.

The Fifth Step, and to me it is tremendously important, is a much neglected one. It requires a bit of time, mental effort, and the application of a rather comprehensive background of knowledge. It is this: Having accumulated all this evidence, sit down with a tabulation of it, study it thoroughly, and then ask yourself these questions: Can it be done in accordance with the accepted morphologic norms? *Should* it be done in compliance with these arbitrary rules? Should it be done at all? Will it benefit the patient or will it do more harm than good? Should it be done now or should some pretreatment regime be instituted? Have I, at my command, the treatment mechanisms or appliances required to do it? These questions, particularly the last one, will require honest, self-effacing inquiry without bias or prejudice. Tabulate all the findings which militate against the successful treatment of the case as you normally would undertake it. Tabulate all the findings which point toward a successful conclusion of conventional treatment. Balance these findings, one column against the other, and then see what the score is. If it is unfavorable, review the evidence, revise your tentative outline, and try again. Continue this investigation until you have a plan, one that will best meet all the known limitations and will best cope with all the probable contingencies which may arise. And remember this. That this requires thought is evident when it is recognized that, disregarding appliances, there are at least six different solutions to the typical Class II malocclusion.

If the orthodontists had a prayer they could do no better than to accept in paraphrase the supplication of one of our Pacific Fleet admirals, "Give us the courage to accept with serenity that which cannot be changed, give us the strength to change that which can and should be changed, and give us the wisdom to distinguish the one from the other."

The Sixth Step.—Select, design, and mentally test the appliance which you intend to use. If it fulfills the requirements of the case, go ahead, you are on ground as safe as can be found for interfering with the face, happiness, and future dentition of a fellow human being. If your appliance is incapable of bringing about the alterations which are required in the exercise of good judgment, change the appliance to meet the needs rather than the treatment outline to fit the limitations of the appliance. Maybe that will require more work.

Maybe a laboratory technician will be unable to construct it. Maybe the end result will be the type of appliance that cannot be adjusted by meeting the child in the reception room, slapping him on the back, and telling him to return in a couple of months. Or maybe I am wrong and all this complex procedure is "old country." It has been said, frequently, that it is, but I am not convinced.

There it is, gentlemen. Controversial, contentious, and perhaps even a little venomous, it is but a *very* small fragment of the thoughts which have been fermenting in my mind for several years and which your deluded program committee insisted you wanted to hear. If I have alienated any of my friends, I am genuinely sorry. Nevertheless, there are things to be said, and when it fell to my lot to say them, there seemed to be no way of extricating myself. For your consolation I offer this proposal. I now shall resume the sackcloth and ashes of the recluse, crawl back into that comfortable sack of oblivion, and remain there permanently, in silence.

Editorial

Orthodontics at the University of Pennsylvania

GRADUATE studies in orthodontics have been a much mooted subject for many, many years. Graduate work in the early days was for the most part done in private schools such as the Angle School, the Dewey School, and others. The Angle School not only pioneered in orthodontic graduate work, but it also supplied a burst of enthusiasm and inspiration to its students that has been nothing short of amazing during the entire history of the specialty. In reality, the Angle School provided the nucleus for the origin of the specialty of orthodontics.

The days of the private schools, however, are now past for the most part and the universities have taken over. The universities increased the length of courses to a year's duration, made them more comprehensive, and have done good work with a difficult subject to teach.

It is interesting now to learn that as a further step in orthodontic education, the Graduate School of Medicine of the University of Pennsylvania, established in 1916 for the purpose of providing graduate medical studies in clinical fields, has enlarged the scope of activities to include graduate studies in dentistry, which will include a comprehensive course in orthodontics under the direction of Dr. John W. Ross of the dental faculty of the University of Pennsylvania.

There will be graduate dental studies in oral surgery, orthodontics, oral medicine, periodontia, and prosthetic dentistry.

There will be a basic period; then the second period of studies must cover two or more years and may occur anywhere that an approved arrangement becomes established, according to the plan.

That which is new and of particular interest is the fact that the dental studies are arranged in three periods, each consisting of a complete unit but leading to the succeeding one. The basic or first period of studies consists of a full academic year and must be taken at the University. Academic credit is accorded for the successful completion of the basic studies and a certificate is awarded. The second period must be preceded by the basic studies at the University but may occur anywhere, provided the specific plan for each candidate receives due approval by the University. This period of studies leads to the degree of Master of Dental Science (M.Sc. [Dent.]). The third period of studies is intended, primarily, to be devoted to research. The location may be anywhere that an approved arrangement becomes established. This period of studies leads to the degree of Doctor of Dental Science (D.Sc. [Dent.]).

The course in orthodontics is designed "to give an understanding of the principles and practice of orthodontics; to study occlusion of the teeth and the development of the face as a problem in evolution; to approach malocclusion of the teeth as a condition associated with other physical growth defects; to develop a scientific biologic approach to orthodontics (the associated medical groups in the Graduate School of Medicine made this approach possible); to consider each individual patient as a problem in etiology based upon a complete

physical and oral examination of the patient; to study normal development at different age levels and to differentiate the normal from the variation leading into abnormalities requiring treatment; to present growth principles underlying all treatment; and to treat cases (by students) using all generally accepted, and special, orthodontic appliances under the direction of a clinical staff of teachers expert in the different technical procedures."

It is obvious that in the teaching of orthodontics in the past there has been a tendency on the part of students to regard the treatment of malocclusion as the routine manipulation of a mechanical ensemble, which some have called "gadget-mindedness," or a tendency at least to discount the importance of the all-important biologic attribute of nutrition and growth. It is to be presumed that with the inclusion of a good measure of the basic sciences in orthodontic education this will prove to be another advance in the step-by-step progress of orthodontic education, and that it will help to indoctrinate the student with an over-all fundamental background of the problem in hand.

Traditionally, orthodontic education has been a difficult subject to handle from an educational standpoint, particularly so since proper treatment of it requires the element of considerable time. Regardless then of basic and theoretical training, the student in the past has been limited in clinical experience. Orthodontic skill like that in other specialties requires wide experience and it has been thought by many that the educational problem will not be satisfactorily solved until some kind of plan can be worked out whereby the prospective specialist in orthodontics can serve an internship that will provide him with ample experience in his chosen field before he is launched into practice.

It would appear that the University of Pennsylvania plan is a step forward in orthodontic education and that it is a courageous effort to make orthodontic training and education as broad and comprehensive as it rightfully should be.

The plan is a convincing demonstration of the ability of large universities to give leadership to graduate educational plans which are desirable and commendable but which cannot gain any momentum without the kind of support which only a large educational institution can provide.

Orthodontics is entitled to broad basic educational advantages because, for the most part, orthodontic undergraduate education is little more than a gesture, through no fault of anyone in particular. Dental schools have neither the time allotment nor the facilities to teach the subject as it should be taught.

H. C. P.

Thumbs Up

AN ARTICLE appeared in the April, 1945, issue of the *Woman's Home Companion* entitled "Thumbs Up" by Dr. Lauretta Bender. Many letters came to the Editor of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY manifesting amazement at the general trend of the article.

Inspired by these letters, the Editor wrote to an orthodontist, Dr. Samuel J. Lewis of Detroit, who has contributed manuscripts on this subject to the JOURNAL on several different occasions in the past. The reply of Dr. Lewis will be of interest to orthodontists everywhere. Dr. Lewis' comments follow:

Dear Doctor Pollock:

In answer to your letter of inquiry dated April 4, 1945, I have just finished reading the article "Thumbs Up" by Dr. Lauretta Bender, in the April issue of the *Woman's Home Companion*.

In my opinion the article contains some truths, some half truths, and some dangerous errors. Where Dr. Bender got her information I do not know, but since she is a child psychiatrist she certainly has access to the best information available on the subject. What she does with it is another story.

When I first made my study of thumb-sucking at the Merrill-Palmer School, in 1924, I, like many of my colleagues, regarded the subject from a purely mechanical and, so to speak, orthodontic point of view. It was evident that thumb-sucking caused teeth to protrude; hence the habit should be broken, regardless of consequences of a psychological nature. Since that time, some of us have sought the advice and counsel of workers in other fields—preschool educators, pediatricians, psychiatrists, and clinical psychologists. Pooling our knowledge and interests, and at the same time adopting an open-minded attitude, we found a common ground upon which to proceed.

There was no question in our minds but that in certain types of cases thumb-sucking caused a protrusion of the upper incisors and at times a retrusion of the lower incisors. However, we found that in most cases, when the habit was broken, the coincidental malocclusion disappeared normally, even as late as the twelfth year. In most cases the child drops the habit by the fourth year.

Thumb-sucking is a normal behavior pattern in babies brought up under *modern civilized conditions*. The habit is almost nonexistent in primitive children who are fed when hungry and not allowed to cry and who are moreover often allowed to suckle until they are 3 years old. Lack of sufficient sucking movements seems to be the most satisfactory explanation of the habit in the great majority of cases. Four-hour feeders suck their thumbs more often than three-hour feeders, and those on no particular schedule suck their thumbs less than the three-hour feeders. Many pediatricians of my acquaintance have adopted what they call the *"hunger schedule,"* that is, they advise the mother to feed the child whenever it evinces hunger by crying, and they report that these babies seldom suck their thumbs. Hence, the advice of Dr. Bender—to encourage thumb-sucking and to help the baby who has trouble in getting his thumb into his mouth—seems to me to be a bit of foolish and unscientific procedure. Better to inquire into the feeding habits of these babies, in my opinion.

We find that, in most children who grow and develop normally and adjust themselves socially, the thumb-sucking habit is dropped by the fourth year. This was true of the Dionne quintuplets, all of whom sucked their thumbs as babies. Four of them dropped the habit when they were about three years old. Emily continued, and the dentist recommended a deterrent appliance. However, the psychiatrist reasoned that since the others had broken themselves of the habit Emily should be given another six months or so of grace, and within this period she too dropped the habit without the assistance of deterrent treatment.

If the habit persists after the sixth year it is no longer to be regarded as accessory to the taking of nourishment but rather as the retention of an infantile pattern. It then becomes a problem, not only for the orthodontist but for the psychiarist as well.

In my own pracice I do not pay much attention to thumb-sucking in babies except to look into their feeding habits and consult with the pediatrician when necessary. The cause of most malocclusions lies much deeper than a local habit, and in my opinion orthodontists lay too much stress on this normal behavior pattern. I never advise mechanical appliances to check the habit, for they do not get at the cause. Moreover, it has been my experience and that of others as well that the child who is prevented from sucking his thumb often takes to masturbation and other undesirable habits. Thus the substitution may lead to consequences far worse than the habit itself.

(Signed) SAMUEL J. LEWIS.

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Max E. Ernst, Secretary, American Association of Orthodontists, 1250 Lowry Medical Arts Bldg., St. Paul, Minn.

Department of Orthodontic Abstracts and Reviews

Edited by

DR. J. A. SALZMANN, NEW YORK CITY

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City

Perlèche: Its Nosologic Status, Chairman's Address, Clark W. Finnerud, M.D., Chicago, *J. A. M. A.* **126**: 737-740, Nov. 18, 1944.

Perlèche manifestations are essentially the same in children and adults, irrespective of etiologic factors. The subjective symptoms of perlèche are mild, usually consisting merely of a feeling of dryness and at times a slight burning sensation. Deep, infected fissures may be painful and, though rarely encountered, occur chiefly in perlèche of adults.

While it is true that in relatively subclinical states of hypovitaminosis the findings may be limited to fissuring or even milder involvement of the oral commissures, in the vast majority of patients with vitamin B complex deficiency other signs of such deficiency are demonstrable.

Objectively, one sees the epithelium of one or both labial commissures early to be mother-of-pearl colored, somewhat macerated, either adherent or easily detached, and wrinkled, later the wrinkles becoming deeper, often forming usually one, sometimes multiple, transverse fissures with red bases, these fissures having little if any tendency to bleed. The involvement usually stops rather abruptly just within the mucocutaneous junction of the commissure but extends as a localized erythematous scaling dermatitis, usually with fissuring, from a few millimeters to as much as 2 or more centimeters outward and downward from the mouth angle onto the skin. Inflammatory changes may extend from the commissures toward the middle of the lips for a centimeter or more, involving chiefly the lower lip. Untreated, the lesions in all types of perlèche have a tendency toward spontaneous remission and exacerbation, but they rarely disappear completely.

It was long considered a disease entity of streptococcic origin. The disorder has also been attributed to staphylococci, chiefly *Staphylococcus cereus.*

We have all encountered treatment-resistant chronic fissuring of the labial commissures, especially in elderly persons, the majority of whom have false teeth. These cases include those instances primarily of mechanical origin, not only those resulting from malocclusion caused by ill-fitting dentures, but also those resulting from the sagging of tissues of elderly persons, general lowered resistance, and salivation induced by dentures.

Under the title "Pseudo Ariboflavinosis," Ellenberg and Pollack carefully studied a group of 34 patients with what they termed perlèche, all except one of whom were 40 to 72 years of age, some of them having an associated glossitis, none of whom had other signs of avitaminosis and none of whom responded to intensive riboflavin and other vitamin therapy. Thirty-two of the 34 patients had upper and lower artificial, ill-fitting dentures, which resulted in a mechanical defect. Their other two patients with perlèche, who did not have false teeth, had mechanical defects to explain the lesions: one, a woman aged 26 years, who presented malocclusion, and the other, an edentulous man aged 72 years, whose upper lip sagged so as to overlie the lower. They were able to demonstrate that the lesions in their cases were not due to specific allergy to the components of the dentures or to the presence of impurities in "undercured" vulcanite plates, but definitely resulted from an improper too short vertical dimension. As a result of the vertical dimen-

sion being too small, overclosure results, the upper lip overlapping the lower, thus producing creases at the corners of the mouth and sagging. They concluded that thus a pocket formation occurs at the lateral angles of the lips, a receptacle for saliva which becomes a medium for fungous growth, and that these fungi are a factor in the production of the cutaneous lesion of perlèche.

Usually perlèche lesions do not appear until after dentures have been worn for as much as several years, during which time bone resorption is known to take place. The mechanical defect, causing a shortened vertical dimension with overclosure, has been concluded by several observers to result most often from ill-fitting dentures, treatment for which consists of correction of the malocclusion.

Symptoms identical to those long recognized as those of perlèche often result in human subjects from riboflavin deficiency, manifestations formerly described as pellagra sine pellagra. The lesions "began as a pallor of the mucosa of the lip in the angles of the mouth without involvement of the buccal mucosa. This pallor was soon followed by maceration, and within a few days superficial transverse fissures appeared, usually bilateral and exactly in the angles of the mouth. These fissures extended somewhat downward from the angle and there was very little inflammatory reaction. The lesions remained moist and became covered with a honey-colored crust which could be scraped off without bleeding. In some instances the fissures extended onto the skin for a distance of as much as ½ inch. The lesions resemble those described as perlèche. At about the time the fissures were seen, the lips became abnormally red along the lines of closure. This was apparently due to a superficial denudation of the mucosa. In some cases, in addition to what was termed cheilosis, there was also seen a fine, scaly, slightly greasy desquamation on a mildly erythematous base in the nasolabial folds, on the alae nasi, in the vestibule of the nose and on the ears."

The manifestations of ariboflavinosis consist of cheilosis, the reddened macerated areas at the angles of the mouth and the linear lesions, and the ocular symptoms, characterized by bulbar conjunctivitis, lacrimation, burning of the eyes and failing vision. Commonly the children were underweight and underdeveloped for their age. Some with advanced lesions of the eyes had no involvement of the mouth angles, whereas, when the eye lesions were slight, bacteriologic study of the fissures of the mouth angles of 93 of the children showed either pure or nearly pure cultures of hemolytic *Staphylococcus aureus* in 80 per cent of the cases, and *Streptococcus hemolyticus* was the predominating organism in the remaining 20 per cent of the cases. Hemolytic strains of *Staphylococcus aureus* were isolated in 14 of 30 cases with conjunctivitis. They found the disease to be noncontagious. Under the simple treatment of 1 mg. of riboflavin three times daily or 1 ounce of brewers' yeast or liver extract daily, the lesions of the mouth angles healed and the ocular manifestations disappeared, and it became impossible to demonstrate the presence of the microorganisms which were present before therapy.

Perlèche manifestations in vitamin deficiency diseases have in rare instances been eradicated by nicotinic acid, after failure with riboflavin, and in the cases reported by Smith and Martin by administrations of pyridoxine.

The heading "pseudoperlèche" might well be included to denote the instances of contact dermatitis, neurodermatitis, seborrheic dermatitis, atrophic dermatitis, severe "chapping," herpes, and so on, which, because of their localization being such as to include the region of the oral commissures, and because of the movement in this area by mastication, talking, and the like, cause a perlèchelike fissuring, merely instances of the inflammatory skin of the respective disorder being so filled with fluid that it can no longer stretch and therefore breaks, just as occurs elsewhere in inflammatory disorders in anatomic locations subjected to movement. It should be remembered that perlèchelike involvement is also seen at times in the rhagades of congenital syphilis and as an extension to this area from mucous patches of syphilis in the mouth. Transitorially, perlèchelike involvement may result from the trauma of manipulations incidental to dental therapy.

All microorganisms claimed to date as being etiological of perlèche have been repeatedly demonstrated as present in several large series of cases of this fissuring intertrigo of the oral commissures in ariboflavinosis and other hypovitaminotic processes, and in mechanical defects resulting from malocclusion from ill-fitting dentures and from the sagging tissues of the elderly. On correction of the hypovitaminosis or mechanical defect, the oral commissural fissuring and the organisms completely disappear. The streptococci, monilias, and other microorganisms therefore appear to be secondary factors in these types of perlèche, factors which merely aggravate the process. In time we may learn that the infection present is always a secondary factor.

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NORTHERN SECTION

The meeting of the Northern Section of the Pacific Coast Society of Orthodontists was held at the Medical Dental Building Auditorium, Seattle, June 24, 1945.

Members present: Drs. D. C. MacEwan, E. S. Weyer, Wm. F. Clarke, P. D. Lewis, J. E. Richmond, M. R. Chipman, H. G. Stoffell, E. J. Fraser, Wm. P. McGovern, E. B. Faxon, E. W. Tucker, Wm. Dinham, M. H. Fisher, H. N. Moore, S. B. Hoskin, G. A. Barker.

Dr. Chipman of Spokane presented a paper entitled, "The Time to Treat." This paper, a digest of the opinions of Northwest orthodontists, was very well prepared and presented.

Dr. Elwood B. Faxon was elected Chairman of the Northern Section.

Dr. Henry G. Stoffell was elected Secretary-Treasurer.

CENTRAL SECTION

The meeting of the Central Section of the Pacific Coast Society of Orthodontists was held at the Alexander Hamilton Hotel, San Francisco, Tuesday, June 12, 1945.

Members present: Drs. Vernon L. Hunt, Arthur F. Skaife, Frederick T. West, J. Camp Dean, Leland E. Carter, Will Gross Sheffer, Reuben L. Blake, Ray A. Lussier, Earl F. Lussier, Charles Konigsberg, Ernest L. Johnson, Allen E. Scott, Thomas R. Sweet, Clarence W. Carey, William S. Smith, Kenneth F. Terwilliger, J. Kester Diment, Bernard Matzen, E. R. Schroeder, Reid Van Noate, Lyle Dale Russell, Fred Wolfsohn.

The guest speaker of the evening was Dr. Wendell Wylie, whose subject was "The Closed Bite; Inherent Factors Governing the Etiology, Treatment and Prognosis." The importance of the physiologic rest position in relation to centric occlusion, muscle tolerance, and individual tolerance were interwoven into both the orthodontic and prosthetic problems as a means of determining how successful the opening of a bite might be.

Subjects and essayists later presented were as follows:

"The Orthodontic Aspect of Dental Medicine." Dr. Hermann Becks.

"Diagnostic and Treatment Records." Dr. Will Sheffer.

"Economic and Financial Outlook." Dr. Dillon T. Stevens.

"A Survey of Dr. Broadbent's work, and an Interpretation of Its Implications to Orthodontics." Dr. Wendell L. Wylie.

"When Should Extraction Be Used as an Adjunct to Orthodontic Therapy?" Dr. Matt Lasher.

"The Etiology of Deficient Dento-Facial Growth." Dr. Wendell Wylie.

"Influence of the Muscles of Mastication on the Development of the Oral and Nasal Cavities." Dr. J. Walter Reeves.

"Some Simple Methods of Treatment." Dr. Eugene I. Gould.

"Roentgenography in Orthodontics" (Pathogenic signs, interpretation, evaluation, etc.). Dr. Hermann Becks.

Symposium—"The Appliance of My Choice and the Reasons Why I Employ It."

Dr. Spencer Atkinson	The "Universal"
Dr. Jess Linn	The "Ribbon Arch"
Dr. James McCoy	The "Open Tube"
Dr. Hays Nance	The "Edgewise"

Drs. Lasher, Linn, McCoy, and Nance discussed the problem of extractions in orthodontic therapy.

The argument covered all angles on the subject, and each member present will be able to adapt the problem to his practice with more confidence; whether it be extractions or no extractions.

Drs. Nance, McCoy, Linn and Atkinson staged a symposium that, a decade ago, would have been a "wonder of wonders." Each came through nobly.

Eugene Gould believes in simple methods of orthodontic therapy and demonstrated several types which have been used to good advantage in his practice.

A comment of the editor of the Central Section reads as follows:

"With the Southern boys not too unanimous on the extraction question we guess many of us will continue with clear consciences extracting only when necessary and not creating too many bi-maxillary protrusions."

Navy Mobile Dental Unit Takes the Dentist to the Patient

If a sailor can't get to a dentist, the dentist goes to the sailor—and takes the best of equipment along in order to give him the finest of treatment.

That's the purpose of the Navy's new mobile dental units, housed in huge trailer trucks, nine of which will be in operation in various sections of the United States by the end of this month. One already is in use in the Third Naval District, which comprises New York, Connecticut, and the northern part of New Jersey, and a second has been assigned to this area. These two units will handle the dental needs of an estimated 13,000 Navy men and women in this district who are at stations where there is no Navy dental treatment or where the facilities are inadequate.

(Official U. S. Navy Photo.)

These 14-ton units, consisting of truck and trailer and costing $22,000 fully equipped, were conceived by Rear Admiral Alexander G. Lyle (Dental Corps) U.S.N., who is the Navy's dental chief. The actual engineering and supervision of construction were detailed to Commander A. W. Borsum (DC) U.S.N., of the Planning Division of the Navy's Bureau of Medicine and Surgery.

Aboard each unit is equipment that measures up to high Navy professional standards. Each has two complete operating units, x-ray equipment complete with darkroom, and steriliz-

ing equipment. The trailers are completely air-conditioned—an important factor because heat and high humidity prevent insertion of good fillings in anterior teeth in hot weather.

There's an interesting sidelight in connection with the equipment aboard the first of the Third Naval District units. It was donated to the Navy by the Dental Society of the State of New York, whose members saved scrap gold—$4,000 worth—to pay the bill.

Local water and power lines may be connected to the units. If they are not available, the units carry a jeep engine which runs the generator which in turn powers the x-ray machine, water and air heaters, sterilizer, air compressor, all lights, and the operating units with their cauteries, warm air and water syringes, aspirators, engines, and electric diagnostic instruments. Each unit carries a tank filled with 120 gallons of water.

Under the direction of Captain Clemens V. Rault (DC) U.S.N., district dental officer, visits to naval stations are scheduled. A unit stays in one spot till the job is done. Typical assignments are to V-12 colleges in upstate New York, to the U. S. Naval Magazine at Montauk Point, to small stations in New Jersey and Connecticut.

In addition, the units will do a great deal of waterfront work—pulling up alongside ships at New York piers to handle the dental requirements of officers and men.

Captain Rault is enthusiastic about the work that can be done with these new units. Supplementing dental installations at large stations and aboard large ships, it is expected that these units will do much to improve dental health of Navy men and women.

The two Third Naval District units are under command of officers who formerly were New York dentists and who have recently returned from tours of sea duty. One is in charge of Lt. Comdr. Richard J. Warnecke (DC) U.S.N., of 693 St. Johns Place, Brooklyn, who entered the service in January of 1942 and has just come back from the Pacific after twenty months aboard an aircraft carrier. The other is under the command of Lt. Harold Y. D. Bonsole (DC) U.S.N.R., of 357 West 55th Street, New York City, who was commissioned in January of 1943 and has been serving in the Atlantic aboard an attack transport.

Each is assisted by another dental officer and two dental technicians, both hospital corps men. The fifth man in each "ship's company" is the driver.

Ten Dental Officers Awarded Bronze Star

Announcement has been made of the awarding of a Bronze Star to the following ten dental officers: Captain William C. Johnson, 85th Infantry Division; Captain Milton W. Barry, 34th Infantry Division; Captain Ira L. Berc, 88th Infantry Division; Major Frank G. Bolton, 85th Infantry Division; Captain Chester J. Dau, VI Corps; Captain Hugo Fielschmidt, 45th Infantry Division; Captain Henry A. Gill, 36th Infantry Division; Captain Theodore Hosbrouck, 85th Infantry Division; Captain Arthur Klein, VI Corps; Captain Leon F. Pellettieri, 34th Infantry Division.

The American Society for the Advancement of General Anesthesia in Dentistry

The fall meeting of the American Society for the Advancement of General Anesthesia in Dentistry will be held on Monday evening, Oct. 22, 1945, at the Hotel McAlpin, Broadway and 34th Street, New York, N. Y. The speakers will be Dr. Nash C. Brennan of Shenandoah, Pennsylvania, and Major Seymour Schotz, M.C., A.A.F., Regional Hospital, Maxwell Field, Alabama.

M. Hillel Feldman, Executive Secretary, 730 Fifth Avenue, New York, N. Y.

Note of Interest

Dr. James S. Hoffer wishes to announce that he has been released from active duty with the Armed Forces and has resumed his orthodontic practice at 1113 Equitable Building, Des Moines, Iowa.

OFFICERS OF ORTHODONTIC SOCIETIES*

American Association of Orthodontists

President, Archie B. Brusse - - - - - - - - - - 1558 Humboldt St., Denver, Colo.
President-Elect, Earl G. Jones - - - - - - - - - 185 East State St., Columbus, Ohio
Vice-President, Will G. Sheffer - - - - - - - - Medico-Dental Bldg., San Jose, Calif.
Secretary-Treasurer, Max E. Ernst - - - 1250 Lowry Medical Arts Bldg., St. Paul, Minn.

Central Section of the American Association of Orthodontists

President, Arthur C. Rohde - - - - - - - - 324 E. Washington Ave., Milwaukee, Wis.
Secretary-Treasurer, L. B. Higley - - - - - - - 705 S. Summit St., Iowa City, Iowa

Great Lakes Society of Orthodontists

President, Willard A. Gray - - - - - - - - - Medical Arts Bldg., Rochester, N. Y.
Secretary-Treasurer, C. Edward Martinek - - - - - 661 Fisher Bldg., Detroit, Mich.

New York Society of Orthodontists

President, Raymond L. Webster - - - - - - - - - 155 Angell St., Providence, R. I.
Secretary-Treasurer, Norman L. Hillyer - - - - - Professional Bldg., Hempstead, N. Y.

Pacific Coast Society of Orthodontists

President, J. Camp Dean - - - - - - - - - - - 1624 Franklin St., Oakland, Calif.
Secretary-Treasurer, Earl F. Lussier - - - - - - 450 Sutter St., San Francisco, Calif.

Rocky Mountain Society of Orthodontists

President, Henry F. Hoffman - - - - - - - - - 700 Majestic Bldg., Denver, Colo.
Secretary-Treasurer, George H. Siersma - - - - - 1232 Republic Bldg., Denver, Colo.

Southern Society of Orthodontists

President, Amos S. Bumgardner - - - - - - - - Professional Bldg., Charlotte, N. C.
Secretary-Treasurer, Leland T. Daniel - - - - - 407-8 Exchange Bldg., Orlando, Fla.

Southwestern Society of Orthodontists

President, Harry Sorrels - - - - - - - - - Medical Arts Bldg., Oklahoma City, Okla.
Secretary-Treasurer, James O. Bailey - - - - - Hamilton Bldg., Wichita Falls, Texas

American Board of Orthodontics

President, Frederic T. Murlless, Jr. - - - - - - - 43 Farmington Ave., Hartford, Conn.
Vice-President, Joseph D. Eby - - - - - - - - - 121 E. 60th St., New York, N. Y.
Secretary, Bernard G. deVries - - - - - - - - Medical Arts Bldg., Minneapolis, Minn.
Treasurer, Oliver W. White - - - - - - - - - 213 David Whitney Bldg., Detroit, Mich.
James D. McCoy - - - - - - - - - 3839 Wilshire Blvd., Los Angeles, Calif.
Claude R. Wood - - - - - - - - - - Medical Arts Bldg., Knoxville, Tenn.
James A. Burrill - - - - - - - - - 25 E. Washington St., Chicago, Ill.

Harvard Society of Orthodontists

President, Francis J. Martin - - - - - - - - - - 1074 Centre St., Newton, Mass.
Secretary-Treasurer, Edward I. Silver - - - - - - - 80 Boylston St., Boston, Mass.

Washington-Baltimore Society of Orthodontists

President, Meyer Eggnatz - - - - - - - - - - Medical Arts Bldg., Baltimore, Md.
Secretary-Treasurer, William Kress - - - - - - - Medical Arts Bldg., Baltimore, Md.

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

St. Louis Society of Orthodontists

President, Virgil A. Kimmey - - - - - - - 3722 Washington Ave., St. Louis 8, Mo.
Vice-President, Leo M. Shanley - - - - - - - 7800 Maryland Ave., Clayton 5, Mo.
Secretary-Treasurer, Everett W. Bedell - - - - 1504 S. Grand Blvd., St. Louis 4, Mo.

Philadelphia Society of Orthodontists

President, John V. Mershon - - - - - - - - 1520 Spruce St., Philadelphia 2, Pa.
Vice-President, Frederick R. Stathers - - - - - - - 269 S. 19th St., Philadelphia, Pa.
Secretary-Treasurer, Augustus L. Wright - - - - 255 S. 17th St., Philadelphia 3, Pa.

Foreign Societies*

British Society for the Study of Orthodontics

President, S. A. Riddett - - - - - - - - - - 42 Harley St., London, W. 1, England
Secretary, R. Cutler - - - - - - - - - 8 Lower Sloane St., London, S.W. 1, England
Treasurer, Harold Chapman - - - - - - 6 Upper Wimpole St., London, W. 1, England

Sociedad de Ortodoncia de Chile

President, Alejandro Manhood - - - - - - - - - - - - Avda. B. O'Higgins 878
Vice-President, Arturo Toriello - - - - - - - - - - - - - - - Calle Londres 63
Secretary, Rafael Huneeus - - - - - - - - - - - - - - - - - Calle Agustinas 1572
Treasurer, Pedro Gandulfo - - - - - - - - - - - - - - - - - - Calle Londres 63

Sociedad Argentina de Ortodoncia

President, Vicente A. Bertini
Secretary, Ludovico E. Kempter
Treasurer, Edmundo G. Locci

Sociedad Peruana de Ortodoncia

President, Augusto Taiman
Vice-President, Ricardo Salazar
Secretary, Carlos Elbers
Treasurer, Gerardo Calderon

Asociación Méxicana de Ortodoncia

President, Guillermo Gamboa - Madero 34-3
Secretary, Rutilio Blanco - Donceles 98-209
Treasurer, Carlos M. Paz - Av. Insurgentes 72

*The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis 5, Mo., U. S. A.